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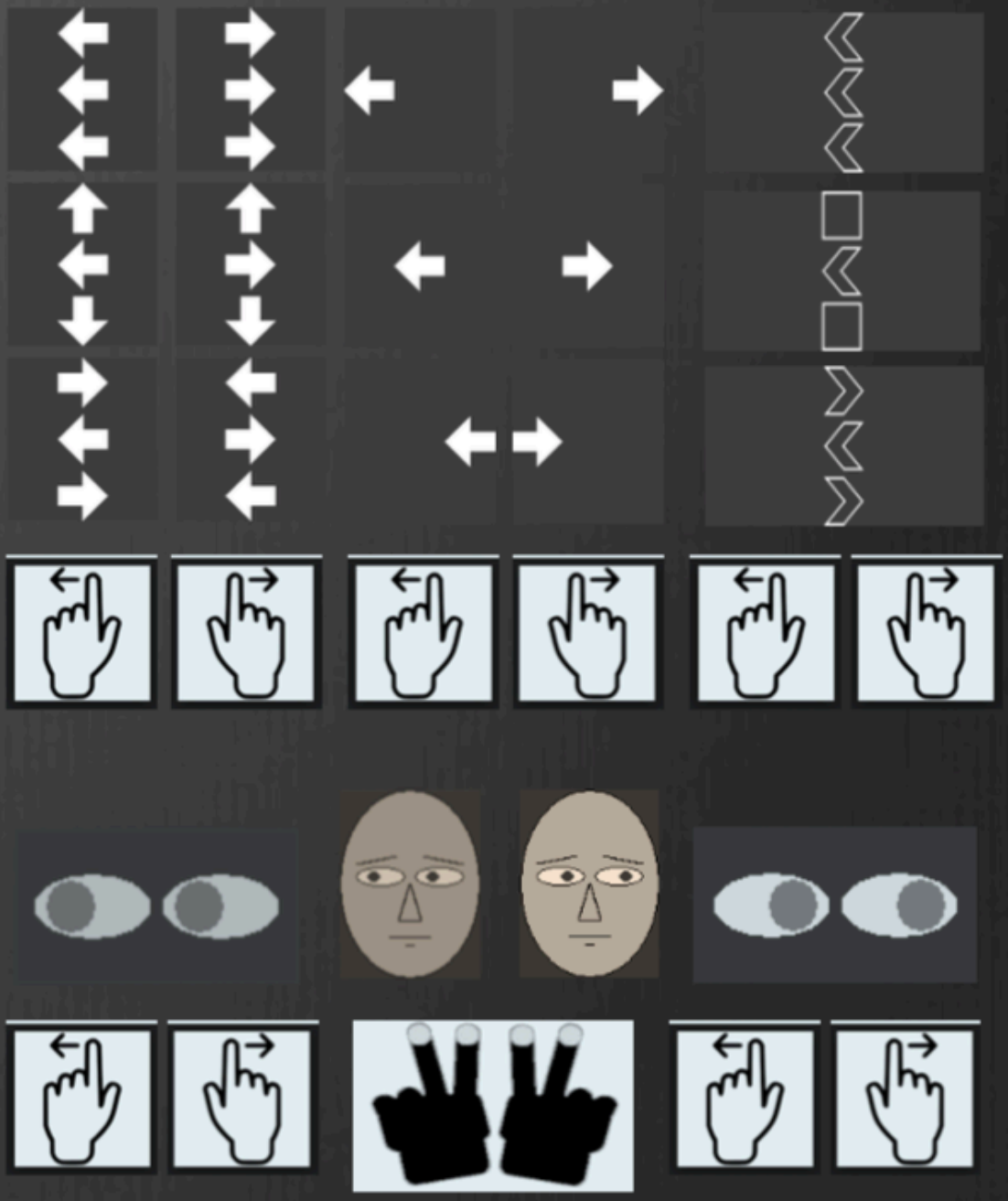

Testing the Flexibility of Cognitive Control using Electrophysiological Correlates of Stimulus-Response Compatibility

PhD Thesis

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Testing the Flexibility of Cognitive Control using Electrophysiological Correlates of Stimulus-Response Compatibility

PROEFSCHRIFT

ter verkrijging van de graad van doctor
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als tijdelijk waarnemer van de functie rector magnificus en uit dien hoofde
vervangend voorzitter van het college voor promoties,
in het openbaar te verdedigen ten overstaan van een
door het college voor promoties aangewezen commissie
in de Aula van de Universiteit
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door

Karen Laura Mansfield
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	Prof. dr. Wim Notebaert
	Prof. dr. Franck Vidal
	Dr. Wouter de Baene

CONTENTS

Chapter 1 (Introduction):
Stimulus-Response Compatibility and the Need for Flexible Cognitive Control

In this thesis I will argue how a clear understanding of stimulus-response compatibility (SRC) is crucial to investigating the flexibility of human cognitive control. In simple terms, SRC refers to the notion that tasks that require us to give a stereotypical or overlearned response upon presentation of a particular stimulus are easier than tasks that require us to give a non-typical response. Traditionally, SRC represents one of the core areas of research into what was once termed "Engineering Psychology", concerned with the relationship between human performance and the machines or systems with which humans interact (see Fitts, 1958). In his 1958 review of Engineering Psychology, Paul Fitts described the available research on stimulus-response compatibility:

> "Investigators continue to find that a highly significant task characteristic is the congruence of stimulus and response patterns. Such factors as correspondence of the direction and planes of motion, the organization of elements, and the linearity or circularity of stimulus and response patterns are found to interact, so that performance is a function of the degree of spatial or of learned correspondence of input and output codes. No general theory has emerged in this area" (Fitts, 1958, p277).

Unsurprisingly, since 1958 several general theories of how SRC influences task performance emerged, many of which are first outlined in this chapter, then put to the test in four electrophysiological studies, and then considered critically in the final chapter. Although nowadays *Engineering Psychology* is an uncommon term, the same areas of research fall under the heading "Human Factors", which extends beyond applied experimental psychology, following a movement in the field toward more cognitive aspects (for a review, see Proctor & Vu, 2010).

On the other side of the same coin, *Cognitive Psychology* is concerned with the limits of human information processing, such as attention and mental workload, and SRC can be used to manipulate the complexity of experimental tasks, increasing mental workload and

the demands on cognitive control. Cognitive psychologists assume that we employ cognitive control to both monitor performance and to facilitate decision-making (Botvinick et al., 2001, Ridderinkhof et al., 2004a, 2004b). Consequently, understanding the interaction between stimulus-response compatibility and cognitive control at the behavioral and neural level is a fundamental topic to both *Human Factors* and *Cognitive Psychology*. Accordingly, this project began as an investigation into the neural correlates of SRC, and developed into an attempt to understand the adaptive and dynamic means by which cognitive control can compensate for varying degrees of task difficulty. Adhering to this path, the rest of this chapter introduces the most important concepts from the SRC literature, then presents the problem regarding the extent to which an account of human decision-making requires the inclusion of control mechanisms, and finally proposes how event related potentials (ERPs) can best be employed to investigate the flexibility of human cognitive control.

1.1 THE ROLE OF DIMENSIONAL OVERLAP

The model of SRC that was most influential to the initial goals of this project is the Dimensional Overlap model (Kornblum et al., 1990; see figure 1.1), which accounts for how the difficulty of a task (or decision) can depend upon the degree of shared features between stimulus and response sets. Kornblum and colleagues expanded on the classic experiments by Fitts (Fitts & Seeger, 1953; Fitts & Deiniger, 1954), who was the first to demonstrate that the difficulty of a task depends not only on the shared features of the stimulus and response sets themselves (dimensional overlap, *set* level SRC), but also on the task/SR-mapping between them (*element* or *task* level SRC). As noted by Kornblum, neither Fitts (1959) nor other researchers who built on his work were consistently accurate in distinguishing these two forms of SRC. However, both Kornblum et al. (1990) and others (e.g. Egner, 2008) have since contributed to a better understanding of the difference between *set* and *task* level SRC. The experiments described in this thesis investigate SRC primarily in relation to compatibility at the *task* level. However, *set* level compatibility is a prerequisite to *task* level SRC, which can best be illustrated by describing the Dimensional Overlap model.

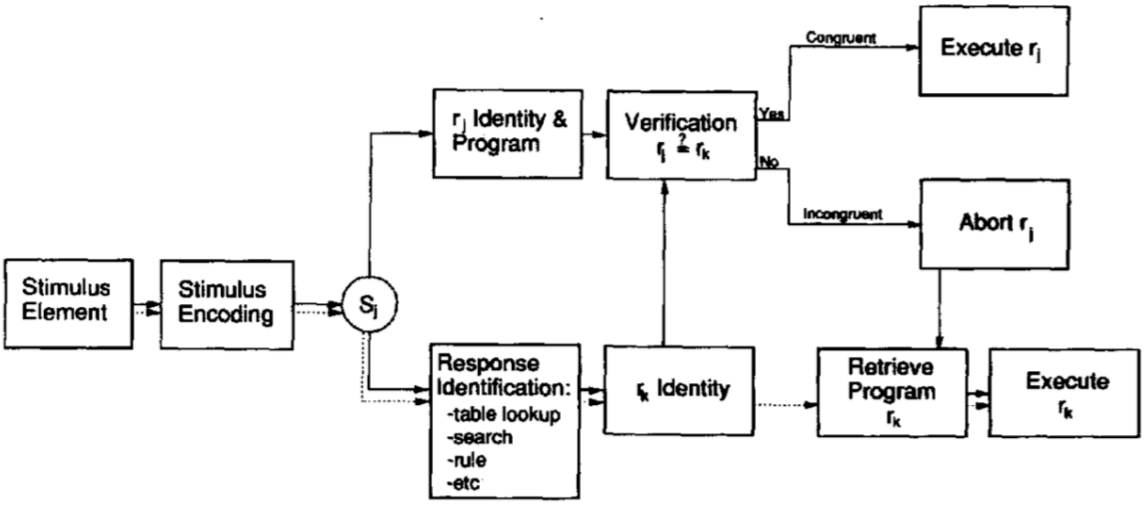

Figure 1.1. The Dimensional Overlap Model (Kornblum et al., 1990), as depicted in figure 3 of the original paper by Kornblum and colleagues. The top route is the direct route of the model, which is automatically active in the case of any dimensional overlap between stimulus and response (in relevant or irrelevant dimensions). The lower route is the indirect route, which requires intentional (controlled) response identification based on only the relevant stimulus dimension. This identity is then used to verify and execute/inhibit the automatic response (if present) and to activate (program) the intended response.

The Dimensional Overlap model defines 3 types of task: *compatible*, *incompatible*, and *noncompatible*. Firstly, any task in which there are no shared features between stimulus and response is assumed to be *noncompatible*, such as when asked to respond to words from 2 different languages with a different-colored response button. *Noncompatibility* refers to SRC at the *set* level, and Fitts & Seeger (1953) were the first to demonstrate its effects in a series of experiments. Secondly, tasks in which the stimulus and response do share features in common (dimensional overlap) could be either *compatible* or *incompatible*. One of the simplest ways to use dimensional overlap to manipulate task incompatibility is in a spatial dimension, such as left vs. right, whereby participants have to decide between responding with the left or the right hand and the stimuli are presented to the left or right of a central fixation point. In this task, when participants are required to give an *in*compatible response (e.g. left stimulus = right-hand response), responses are slower and accuracy levels lower compared to when a spatially compatible response is required (Fitts & Deininger, 1954). Kornblum et al. (1990) described this phenomenon as the "mapping effect", and postulated that the size of the mapping effect could be used as a metric of the degree of compatibility between stimulus and response sets (i.e., to estimate the *set* level SRC).

1.2 RELEVANT VS. IRRELEVANT TASK FEATURES

An additional distinction made by the Dimensional Overlap model is between *relevant* vs. *irrelevant* features of the stimulus in relation to the response decision. The mapping effect is a demonstration of task-*relevant* SRC, because for example the location of the stimulus determines the correct response hand. But consider the task in which a color (e.g. blue vs. green) determines which hand to respond with, but the stimuli are presented either to the left or right of a central fixation point. This is a classic example of the Simon task (Simon & Rudell, 1967), which demonstrates how dimensional overlap between stimulus and response features can lead to interference in response selection even when the shared features are not relevant to the task. Typically, when the irrelevant location of the stimulus corresponds with the location of the response (*congruent* trials), responses are faster and more accurate compared to noncorresponding (*incongruent*) trials (Hommel, 1993; Leuthold & Schröter, 2006; Masaki et al., 2007; Melara et al., 2008; Simon & Rudell, 1967; Wendt et al., 2006).

The final SRC distinction by Kornblum et al. (1990) that is fundamental to this thesis and referred to by Fitts (1959) as S-S compatibility, concerns compatibility between features of the stimuli. A frequently used example of S-S compatibility between *relevant* and *irrelevant* stimulus features is the flanker task (Eriksen and Eriksen, 1974); a central, task-relevant symbol (< = left, > = right) is flanked by irrelevant noise symbols that could be either *congruent* (e.g. >>>>>) or *incongruent* (<<><<) with the central target. Similar to the Simon effect, responses are generally faster and more accurate in *congruent* trials compared to *incongruent* trials (Eriksen & Eriksen, 1974; Kopp et al., 1996; Ridderinkhof et al., 1995; van Veen & Carter, 2002; Wendt et al., 2007). However, in line with the distinction by Kornblum et al. (1990), the flanker and Simon tasks differ in terms of the source of dimensional overlap. This is because in the flanker task the source of interference (the *task-irrelevant* stimulus noise) has dimensional overlap with both the *task-relevant* stimulus features (arrow direction) and the response features (hand); on the contrary, in the Simon effect the *irrelevant* stimulus dimension (location) has dimensional overlap only with the response (hand).

The consequences of the difference between Simon and flanker effects becomes apparent when you consider a flanker task in which participants are asked to give an incompatible response to the central target (e.g. <=right, >=left). In *incongruent* stimulus

displays (e.g. <<><<), the irrelevant flanker noise is *incongruent* with the relevant target but *congruent* with the correct response. In other words, flanker effects might reflect a combination of S-S interference and S-R interference, even with a compatible mapping. Two of the chapters in this thesis investigate the source(s) of interference and its resolution in flanker tasks using ERPs, and these studies were designed to elaborate on a background of theories accounting for SRC effects.

1.3 DUAL-ROUTE MODELS

Accounts of SRC are typically based upon a dual-route model (e.g. de Jong, et al., 1994; Kornblum et al., 1990; Ridderinkhof et al., 1995). A fast, direct-route is assumed to process the stimulus *unconditionally,* inducing automatic response activation by shared features between stimulus and response. A slower, indirect-route is assumed to process the stimulus *conditionally,* involving intentional selection of the relevant features and appropriate response according to the symbolic SR-bindings defined by task instructions. For example, with an incompatible mapping, a stimulus presented on the left will activate a left-hand (incorrect) response quickly via the direct route and will activate a right-hand (correct) response more slowly via the indirect route. Essentially, although it is assumed that task-*relevant* stimulus features are processed via both routes of the model, task-*irrelevant* stimulus features supposedly processed only via the fast direct route. So when any feature of the stimulus is incompatible with the correct response, dual-route models predict interference between two active response options.

Kornblum and colleagues (1990) propose two mechanisms for how the dual-routes of the Dimensional Overlap model can account for the 'Mapping effect". The first mechanism is that the indirect route will take more time to translate the SR bindings with an incompatible mapping than with a compatible mapping. The second mechanism is that the two routes are assumed to result in competing activation at the response level, such that with an incompatible mapping, activation of the compatible (incorrect) response will be detected and inhibited in order to execute an incompatible (correct) response. One of the goals of this project was to test these two key assumptions; by replicating the mapping effect at the behavioral level, while using ERPs to assess the temporal dynamics of competing response

activation, detection of response interference, and inhibition of the incorrect response tendency.

1.4 AUTOMATIC VS. INTENTIONAL PROCESSES

An unclear aspect relating to dual-route models concerns the potential *automaticity* of processing via the indirect route. Strictly, only activation via the direct route takes place *automatically*, and activation via the indirect route takes place *intentionally*, requiring *controlled* processing (c.f. Kornblum et al., 1990; Ridderinkhof et al., 1995). The assumption of the Dimensional Overlap model is that automaticity of response activation and dimensional overlap are synonymous, and are a requirement for processing via the fast, direct route; if there is no dimensional overlap, there will be no compatible response, and only the indirect route will be active. The definition of *automaticity* adopted by Kornblum et al. is close to that of Kahneman and Treisman (1984), and assumes that an *automatic* process cannot be prevented but can be attenuated or enhanced by additional (*intentional*) processing. In other words, in the case of an *incompatible* mapping or *incongruent* stimulus feature, the dimensional overlap model assumes that automatic response activation via the fast direct route will always need to be attenuated, suppressed or inhibited in order to allow the alternative (correct) response to be executed. One of the goals of this project is to use ERPs to assess the functional and temporal properties of such a mechanism, if it exists, for which candidate ERP components are described later in the introduction.

In addition to automatic processing via a direct *unconditional* route, the dual-process model by de Jong and colleagues (1994) proposes automatic *conditional* processing (via an indirect route). Specifically, this proposal aimed to account for the finding of a *reversed* Simon effect: with an incompatible color mapping (e.g. red stimulus = green response button, etc.), de Jong et al. found that participants performed better when the *irrelevant* stimulus location (above vs. below fixation) was *incongruent* with the response location. The same reversal of the Simon effect had been reported previously (Hedge & Marsh, 1975), and similar to the 'logical recoding' account by Hedge & Marsh, de Jong et al interpreted this finding as *automatic generalization* of the mapping reversal rule from the *relevant* dimension (color) to the *irrelevant* dimension (location). The reversal of the Simon effect still poses an interesting challenge, and chapter 3 of this thesis presents possible accounts for

this effect and puts these to the test using ERP measures of response activation and cognitive control.

1.5 AUTOMATICITY VIA FEATURE BINDING

Reversed Simon effects have since been found to depend upon *sequential effects* (alternations vs. repetitions in SR *congruence*) and have been interpreted to reflect mostly repetition priming between SR bindings (Hommel, 2004; Spapé et al., 2011; Wendt et al., 2006). Repetition priming is a form of *automatic* SR translation that adheres to the feature-binding account (Hommel, 2004); shared features between consecutive stimuli (S1 and S2) are assumed to lead to *automatic* reactivation of the same response (R1), even when a different response is required to S2. The feature-binding hypothesis is also grounded on work by Kahneman and colleagues (1992), who demonstrated that being presented with the same stimulus twice in a row facilitated the same response. Kahneman et al. (1992) proposed that when responding to a specific stimulus a temporary, distributed episodic representation is created (called an "object file") that stores and integrates the currently associated features of stimulus and response, thereby facilitating the same response in the event that any of the stimulus features are repeated on the next trial. As such, the complete SR-binding between all features of stimulus and response will have been set up partly by *intentional* processes that relate the task-relevant features of the stimulus to the relevant response, but the irrelevant features of the stimulus will be *automatically* integrated with the task-relevant SR-bindings. Much research has attempted to assess whether feature binding/integration (i.e. *automatic* processing) is enough to account for sequential trial effects, or whether a complete account requires including the role of *control* processes that compensate or correct for such interference. Chapter 4 of this thesis addresses this question, assessing ERP correlates of response activation and cognitive control in relation to sequential trial effects.

1.6 ACCOUNTING FOR SRC EFFECTS IN MORE COMPLEX TASKS

Accounting for performance becomes increasingly challenging with more difficult tasks, suggesting the need to include mechanisms of cognitive control (Logan, 1985). For example, when both compatible and incompatible SR mapping rules are combined in the same experimental block, responses are generally slower and the mapping effect is reduced

(Christensen et al., 2001; de Jong, 1995; Heister & Schroeder-Heister, 1994; Proctor & Vu, 2002; Shaffer, 1965; Stoffels, 1996a, 1996b; van Duren & Sanders, 1988; Vu & Proctor, 2004) or reversed (Jennings et al., 2002), reflecting the greatest performance detriments to compatible mappings (e.g. Stoffels, 1996b, Vu & Proctor, 2004). There are multiple factors that contribute to these effects, and accounts vary regarding the extent to which they reflect cognitive control.

Some have proposed that when compatible and incompatible trials are mixed, automatic response activation (the direct route) is suppressed, particularly following incompatible trials (de Jong, 1995; Duncan, 1978; Kornblum et al., 1990; Praamstra et al., 1999; Ridderinkhof, 2002; Shaffer, 1965; Stoffels, 1996b; Stürmer et al., 2002, 2007; Vu & Proctor, 2004). Such an additional control mechanism can explain slower responses, the reduction to mapping effects, and the specific detriment to compatible trials, which would no longer benefit from automatic response activation. However, suppression of the direct route is not sufficient to account for a reversal to the mapping effect or to the Simon effect, which implies the involvement of alternative processing via an indirect route.

For example, indirect route processing might be compared to the preparation and activation of task schemas (cf. Norman & Shallice, 1986), which represent task procedures in memory. Support for the role of competing task schemas comes from the task-switching paradigm: there are usually costs associated with task-alternations (or task-switches) compared to task-repetitions (for a review see Monsell, 2003). However, task-switching costs are reduced when participants are warned ahead of the upcoming task (see Monsell, 2003; Vandierendonck et al., 2010), which suggests that at least part of the additional processing associated with alternating between SR-bindings (or mapping rules) could be executed prior to presentation of the relevant stimulus.

If a task can be prepared in advance to respond differentially to specific relevant stimuli (c.f. Jennings & van der Molen, 2005), then as soon as a relevant stimulus appears the response might occur relatively automatically. Even for unpracticed or unfamiliar tasks, some have suggested that specific SR-bindings can be set in place as a “prepared reflex” (Cohen-Kdoshay & Meiran, 2009; Hommel, 2000; Woodworth 1938), and more recent research assessing first-trial performance in newly learned tasks (e.g. Cole et al., 2010; Dumontheil et al., 2010) appears to support this idea. The role of preparatory processing is

interesting because it implies that an otherwise difficult task might be executed relatively easily, thereby reducing the demand for cognitive control during online decision-making.

1.7 THE ROLE OF CONTROL

So to what extent can we use preparatory control to facilitate (near) automatic execution of a task? In the most extreme perspective, in line with the idea of a "prepared reflex" (Cohen-Kdoshay & Meiran, 2009; Hommel, 2000; Woodworth 1938), sufficient preparation might allow completely automatic execution of the task. Alternatively, even with careful preparation, additional control might be required to reinforce SR-bindings in the case of too much interference or 'conflict'. In an attempt to solve the problem of the 'homunculcus' (what *instigates* control?), Botvinick and colleagues (2001) proposed that conflict initiates adjustments to cognitive control. Based on both experimental and brain imaging data, and supported by a series of simulations, they developed a 'conflict-monitoring' model; this model proposes a feedback loop such that detection of response conflict (competition between active responses) by anterior cingulate cortex (ACC) leads to control adjustments via pre frontal cortex (PFC). The definition of 'conflict', and assessing the degree of conflict required to instigate an increment to control, usually relies upon simulations (Botvinick et al., 2001; Carter & van Veen, 2007; Yeung et al., 2004). However, analysis of ERPs can provide alternative methods for assessing the degree of response interference and for investigating the potential role of cognitive control, as will be discussed toward the end of this chapter.

1.8 ASSESSING THE FLEXIBILITY OF CONTROL

So far we have discussed the need (or not) for control, the extent to which SR-bindings can be prepared in advance (reducing the need for on-line control), and the relevance of differentiating functional control mechanisms from the degree of response interference (or conflict) present. Providing insight into each of these open topics will need to consider the complexity of the tasks involved. Essentially, complex tasks require not only increased control but also flexible strategies, which should successfully utilize task instructions and other available information.

One recent model that presents cognitive control as being both flexible and adaptive is the Dual Mechanisms of Control (DMC) model (Braver et al., 2007). Braver and colleagues

define preparatory processing as *proactive* control and resolution of conflict as *reactive* control, suggesting 2 potential roles for reactive control: as a signal to increase proactive control on subsequent trials (adaptive behavior) or as a signal to quickly resolve response conflict on the current trial (late correction). Proactive control relies upon predictive contextual information such as that provided in task instructions, which is then used to hold behavioral goals in working memory prior to stimulus presentation, aiming to prevent distraction from irrelevant stimulus features. On the other hand, reactive control is triggered by any response conflict that results following stimulus presentation. The DMC builds on research on working memory function, presumed to rely upon PFC (e.g.. Cohen et al., 1997), with research on conflict detection, assumed to rely upon ACC (Botvinick et al., 2001).

So according to the DMC account (Braver et al., 2007), cognitive control should play an essential, dynamic role in all experimental tasks, from the most simple to the most complex. In the simplest tasks, proactive control should be sufficient to maintain the task goals and associated SR-bindings in working memory, leaving little work for reactive control. However, unpredictable response interference, such as initiated in incongruent trials, can be resolved by reactive control. In more complex tasks, such as the task-switching paradigm, there is an even greater chance of response interference, particularly when switching between tasks. Still it is unclear whether the additional time taken in complex tasks reflects increased *proactive* control and/or increased *reactive* control, or simply the additional time taken to resolve alternations in SR-bindings (c.f. the feature-binding account, Hommel, 2004). With careful manipulation of task demands and response interference, ERPs can be measured to assess the extent to which task-bindings can be prepared effectively and the potential involvement of mechanisms of cognitive control.

Another consideration regarding the flexibility of cognitive control is whether response interference or 'conflict' is detected and resolved by the same underlying mechanisms (e.g. a general conflict monitor, c.f. Botvinick et al., 2001) across different task settings and with different sources of conflict? Egner (2008) refers to this as the question of 'domain-specificity' of conflict resolution. In line with the conflict-monitoring theory, it might be the case that detection of conflict by a *general* conflict-monitor leads to an increase in *general* preparatory control, thereby strengthening currently relevant SR-bindings. On the other hand, different sources of interference (such as irrelevant locations vs. flanker stimuli)

all accumulating at the response level, might be prevented or resolved by different control strategies. Chapters 2 and 3 present and test several potential specific control strategies by comparing Simon and Eriksen interference on performance and ERP data.

1.9 ERP CORRELATES OF SRC AND CONTROL

The next sections introduce the most relevant ERP measures to an investigation of the neural correlates of SRC, cognitive control, and their interaction, using the dimensional overlap model as a reference frame. We can use ERPs associated with (competing) response activation to investigate the neural correlates of SRC; and ERPs associated with detection of (response) interference and selection/inhibition of competing responses to investigate the neural correlates of control. Importantly, we need to consider how SRC and control might interact with increasing task difficulty, and the expected effects on the ERP measures.

1.9.1 ERP Measures of Response Activation

A well-used ERP measure with the potential to assess the degree of automatic response activation when deciding between a left- or right-hand response is the Lateralized Readiness Potential (LRP). The readiness potential describes the increasingly negative-going wave recorded over the motor cortex contralateral to the response hand prior to response execution (Kornhuber & Deecke, 1965). The LRP is a derivative of the readiness potential, subtracting ipsilateral from contralateral motor cortex activity, thereby providing a representation of response activation in terms of correct vs. incorrect response activation (Coles 1989; de Jong et al, 1988; Gratton et al., 1988, 1992). Typically, activation in favor of the responding hand peaks just prior to execution of the response.

Importantly, in LRPs for incongruent flanker trials, an earlier deflection has been detected prior to correct response activation, suggesting automatic activation of the incorrect response (e.g. Gratton et al., 1988, 1992; Heil et al., 2000, Kopp et al., 1996; van ‘t Ent, 2002; Wascher et al., 1999; Willemssen et al., 2004). However, with lateralized stimuli such as in the Simon task, extra care should be taken in interpreting the extent to which early deflections over motor cortices reflect automatic response activation because such activity coincides with much larger deflections recorded over parietal cortices, suggesting contamination of the LRP by visual processing of the lateralized stimulus (Praamstra &

Oostenveld, 2003; Valle-Inclan, 1996; van der Lubbe et al., 2001; Wascher & Wauschkuhn, 1996). Nevertheless, the LRP offers a useful means for investigating the relative amount of activation contralateral vs. ipsilateral to the stimulus and/or response.

More recent studies have investigated motor cortex activation with greater spatial resolution using current source density (a Laplacian transformation), which renders the signal sufficiently sensitive to assess the individual contribution of each hemisphere, and found deflections suggesting inhibition ipsilateral to the response hand with a similar timing to activation contralateral to the response hand (Burle et al, 2004; Carbonnell et al., 2004; Praamstra & Seiss, 2005; van der Laar et al., 2012, 2014; Vidal et al., 2003). Interestingly, the ipsilateral deflection is only found when participants are required to choose between a left- and right- hand response (Burle et al, 2004; Carbonnell et al., 2004), suggesting that it is related to competition between available responses. As such, ipsilateral activity contributes to the pattern found in LRPs, and separate assessment of activity over contra- and ipsi-lateral motor areas can offer an additional dimension for investigating the degree to which increasing task difficulty affects response competition. It has been proposed that the ipsilateral deflection reflects inhibition of the inappropriate response tendency (c.f. Burle et al., 2004), which postulates a helpful tool for investigating the mechanisms of decision-making and response selection in complex tasks.

1.9.2 ERP Measures of Cognitive Control

The ERP component most commonly associated with cognitive control is the fronto-central N2, a negative component peaking shortly after 200ms following stimulus onset (for a review see Folstein & van Petten, 2008). For example, with incongruent flankers, N2 peaks around 300 ms post-stimulus and its amplitude is enhanced relative to congruent trials (e.g. Bartholow et al., 2005; Heil et al., 2000; Kopp et al., 1996; van Veen & Carter, 2002; van ‘t Ent, 2002; Wendt et al., 2007). Midline N2 is also enhanced with incongruent locations in the Simon task (e.g. Carriero et a., 2007; Melara et al., 2008). However, interpretations of N2’s sensitivity to response interference differ. Many studies have interpreted N2 amplitude as a measure of conflict processing (Carter & van Veen, 2007; Donkers & van Boxtel, 2004; Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003; van Veen & Carter, 2002; Yeung et al., 2004), implying that a larger N2 reflects a greater degree of response conflict.

Other studies have interpreted the amplitude of N2 to reflect control processes that resolve response interference, such as suppression of the inappropriate response tendency (Carriero et al., 2007; Falkenstein et al., 1999; Falkenstein et al., 2002; Heil et al., 2000; Kopp et al., 1996; van Boxtel et al., 2001). According to the response-monitoring hypothesis, the amount of conflict and the degree to which control is adjusted should be correlated. This implies that dissociating between the neural mechanisms of these closely-related concepts requires careful experimental manipulation.

Besides incongruent trials, midline N2 is also reported to be sensitive to unexpected events, which are assumed to represent another factor that increases response conflict (Nieuwenhuis et al., 2003). Bartholow and colleagues (2005) compared such 'expectancy-related' conflict with conflict resulting from incompatibility, by manipulating flanker congruency and the probability of incongruent flankers between blocks. They found that N2 was enhanced with incongruent flanker trials, but particularly when incongruent trials were most expected (80% probability); in fact, the sensitivity of N2 to incongruent flankers was only reliable when incongruent trials were highly probable, suggesting that N2 reflects more than the degree of conflict.

In the same study, Bartholow et al. (2005) also investigated the role of response-locked components associated with response conflict: the Ne/ERN, which is enhanced following errors compared to correct responses (Falkenstein et al., 1991; Gehring et al., 1993); and the CRN, which is smaller than the ERN but can be detected following correct responses. Both of these components have been suggested to reflect a response-comparison process (Vidal et al., 2000), particularly in light of their similarity and the sensitivity of CRN to response competition (Scheffers et al., 1996; Vidal et al., 2000). Bartholow et al. (2005), found that response-locked CRN reflected the mismatch between the expected conflict and the actual conflict, such that in each block CRN was largest for the lower probability trials. In other words, CRN but not N2 was consistently associated with the degree of conflict in correct trials, while N2 appeared to reflect control processes that drew on much more than response conflict. Essentially, disentangling the neural mechanisms of cognitive control from response conflict should lend to a better understanding of human decision-making. While brain imaging studies have already provided insights into the topography of conflict detection (by ACC) and control adjustments (via PFC), ERPs could offer a more practical

means to assess the functional, topographical and temporal dynamics of control processes in relation to both task demands and performance.

1.10 USING ERPS TO ASSESS THE INTERACTION BETWEEN SRC AND CONTROL

Returning to the Dimensional Overlap Model, Kornblum et al. (1990) did not define precisely the mechanisms of cognitive control, but the model can be used to illustrate how control strategies might interact with the effects of SRC on decision-making. The DO model (see figure 1.1 at the beginning of this chapter) refers to "controlled" processes that are assumed to 'identify' the correct response, 'verify' that the automatically activated response is the correct one, and then 'execute' or 'inhibit' the activated response. Firstly, the LRP and lateralized ERPs can be used to investigate the degree of automatic response activation in the direct route by task-relevant and task-irrelevant stimulus dimensions, but taking particular care when using lateralized stimuli to control for carry-over from visual processing to motor processing. Subsequently, midline and lateralized ERPs can be used to assess the extent to which increased task demands and response interference are reflected in control mechanisms.

We assume that 'identification' of the correct response via the indirect route should mostly be set up in advance by preparatory processing (or proactive control), but 'verification' of whether the (automatically) activated response is the correct one will require online controlled processing (or reactive control), with or without 'conflict detection'. As such, early ERPs such as midline N2 and ipsilateral positivity are most likely to reflect task difficulty, and to be enhanced by increased preparatory processing (proactive control) in more difficult task blocks. In contrast, later ERPs such as response-locked components (e.g. CRN or pre-response components) should be enhanced by detection and/or resolution of response interference that has not been prevented by preparatory processing, reflecting between-trial variations such as stimulus congruence, compatibility (SR- mapping) and sequential effects.

1.11 GOALS OF THIS THESIS

In sum, there is little doubt that any account of human decision-making needs to include both the role of SRC and the potential mechanisms of cognitive control. What still needs refining is the mechanisms underlying interference by automatic response activation in

different task settings, but particularly the extent to which such interference can be prevented by preparatory (proactive) control, and/or resolved by online (reactive) control. Furthermore, it is relevant to assess the domain-specificity of conflict resolution, in order to elaborate on current theories of conflict monitoring and cognitive control. With increasing task complexity, just how flexible is cognitive control?

Major Research Questions:

1. What are the mechanisms of online reactive control processes involved in resolving unexpected interference between competing responses, and can we use ERPs to differentiate the neural correlates of conflict from functional control mechanisms?
2. To what extent can stimulus-response bindings be prepared in advance to allow automatic responding upon presentation of the stimulus?
3. Are the mechanisms of conflict resolution domain general or domain specific, and which control strategies are most successful with specific types of conflict?

1.12 OVERVIEW OF THE CHAPTERS IN THIS THESIS

Each of the four experimental chapters in this thesis (2-5) aims to address one or more of the major research questions presented above, and chapter 6 is an integrated discussion that aims to answer the three major research questions. The goals of each chapter are outlined below.

Chapter 2: Temporal Dynamics of Simon and Eriksen Interference

Chapter 2 aimed to provide insight into research questions 1 and 3. This experiment (published as Mansfield et al., 2013, Brain & Cognition) used LRPs and lateralized ERPs to assess the automaticity of response activation by irrelevant stimulus features, and midline and lateralized ERPs to assess the domain-specificity of conflict resolution. We compared two different forms of response interference: location (Simon effects) and flankers (Eriksen effects). In line with a dual-route model of SRC, we hypothesized that only Simon interference is incurred via the fast direct route of the DO model, and that flanker interference is incurred via the indirect route. As such, we predicted that Eriksen effects would be larger than Simon effects, and that each type of interference resolution would be reflected in separate control components, supporting the hypothesis of domain-specificity in

conflict and control. We assessed the temporal dynamics of interference by locations and flankers using the LRP and lateralized ERPs, and we assessed the temporal dynamics of cognitive control in conflict resolution using midline and lateralized N2.

Chapter 3: Domain Specificity of Conflict and Control

Chapter 3 attempted to address all three research questions in one experiment. Elaborating on the findings of chapter 2, in which only incongruent locations appeared to incur interference via the fast, direct rout of the DO model, this experiment assessed the potential role of automatic response activation via the indirect route of the model by both irrelevant locations and irrelevant noise (flankers). Additionally, we assessed the nature of preparatory control strategies in preventing response interference by comparing Eriksen and Simon interference (within-blocks) with compatible and incompatible mappings (between-blocks). We aimed to replicate the finding reported in Chapter 2 of larger interference effects for flankers compared to locations, and to test the assumption regarding domain specificity of conflict resolution, assuming that incongruent flanker arrows incur interference via the *indirect* route only, while incongruent locations incur interference via the *direct* route only. As such, we hypothesized a contrasting pattern of effects for flankers/locations in incompatible blocks. Specifically, we predicted that with an incompatible mapping only flanker arrow direction effects and not stimulus location effects would be reversed with the mapping instruction, such that irrelevant locations would always facilitate the corresponding response, but irrelevant flanker arrows would facilitate the opposite response to the arrows' direction. We assessed response interference using LRPs and lateralized ERPs, and we assessed cognitive control involved in conflict resolution using midline and lateralized N2.

Chapter 4: Proactive and Reactive Control in Stimulus-Response Compatibility

Chapter 4 addressed research questions 1 and 2. This study (published in Psychophysiology, Mansfield et al., 2012) investigated the dynamic interplay between proactive and reactive control by assessing behavioral and ERP data when tasks are mixed (two SR-mappings) compared to when tasks are blocked (just one mapping). In order to manipulate SR-mapping within blocks in the left/right spatial dimension with strong dimensional overlap, but without incurring carry-over effects from visual processing, we used gaze stimuli that were directed either left or right with two levels of eccentricity: moderately left/right or extremely

left/right. In line with the difficulty of mixed mapping tasks, we predicted that mixed SR-mapping tasks (in which participants were required to apply an incompatible mapping to just one level of eccentricity, counterbalanced within-participants) would demonstrate: enhanced proactive control, involved in maintaining two SR-mappings in working memory and potentially suppression of the direct route; enhanced reactive control on SR-mapping alternations, involved in late correction of SR-bindings; and potentially a bias toward the reversed (most difficult) mapping. We reasoned that proactive control would be reflected in early stimulus-locked components and that reactive control would be reflected in later response-locked components, in line with the slower responses that can be expected in the mixed SR-mapping task. We assessed response interference using LRPs, proactive control using midline stimulus-locked N2, and reactive control using midline and lateralized response-locked components.

Chapter 5: Testing the Interplay between Proactive and Reactive Control

Chapter 5 addressed research questions 1 and 2. This study used behavioral and ERP measures to assess the dynamic interplay between proactive and reactive cognitive control by comparing predictable and unpredictable situations. This study aimed to replicate the behavioral and ERP findings relating to reactive control reported in Chapter 4, and subsequently to assess whether preparatory (proactive) control is able to reduce the load on reactive control depending on the predictability of the S-R mapping. Using left/right gaze stimuli in green/blue (color signaling the relevant SR-mapping), we used three mixed SR-mapping tasks in which the probability of compatible/incompatible SR-mappings was either 80/20%, 50/50%, or 20/80%. We hypothesized that increased predictability in the SR-mapping would lead to reduced reactive control measures for the 'expected' SR-mapping and increased reactive control measures for the 'unexpected' SR-mapping, compared to 50/50% (unpredictable) blocks. In addition to assessing the behavioral and ERP measures used in chapter 4, we also assessed laplacian transformed lateralized ERPs over motor areas ipsilateral and contralateral to the responding hand, in order to investigate the potential role of inhibition of the spatially corresponding response in both predictable and unpredictable situations.

Chapter 6: Just How Flexible is Cognitive Control?

The final chapter aims to relate the findings of the experimental chapters back to the initial research questions and overall goals. After summarizing the findings of each experimental chapter individually, a higher-level general discussion aims to identify the extent to which the studies in this thesis offered insight into each major research question. This is achieved by first integrating the findings in terms of the initial hypotheses and closely-related background literature, and subsequently by broadening the discussion to consider other methods and lines of research that have attempted to answer one or more of the research questions from a different approach. A concluding section integrates the discussion of all three research questions to consider the extent to which both the findings in this thesis and the recent literature can already shed light on the flexibility of cognitive control, addresses the limitations of this thesis, highlights unanswered questions, and offers suggestions for future research.

Chapter 2:

Temporal dynamics of interference in Simon and Eriksen tasks considered within the context of a dual-process model

Behavioral and brain potential measures were employed to compare interference in Eriksen and Simon tasks. Assuming a dual-process model of interference elicited in speeded response tasks, we hypothesized that only lateralized stimuli in the Simon task induce fast S-R priming via direct unconditional processes, while Eriksen interference effects are induced later via indirect conditional processes. Delays to responses for incongruent trials were indeed larger in the Eriksen than in the Simon task. Only lateralized stimuli in the Simon task elicited early S-R priming, maximal at parietal areas. Incongruent flankers in the Eriksen task elicited interference later, visible as a lateralized N2. Eriksen interference also elicited an additional component (N350), which accounted for the larger behavioral interference effects in the Eriksen task. The findings suggest that interference and its resolution involve different processes for Simon and Eriksen tasks.

Chapter 2 is published in *Brain and Cognition*:
Mansfield, K. L., van der Molen, M. W., Falkenstein, M., & van Boxtel, G. J. (2013). Temporal dynamics of interference in Simon and Eriksen tasks considered within the context of a dual-process model. *Brain and Cognition, 82*(3), 353-363.

2.1 INTRODUCTION

Over the last decade, detailed theories have been developed to account for how cognitive control prevents and amends errors based on conflict detection (Carter & van Veen, 2007; Botvinick, Braver, Carter, Barch, & Cohen, 2001; van Veen & Carter, 2002; Yeung, Botvinick, & Cohen, 2004; for a review see Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004). Conflict-monitoring theories assume a feedback loop that enforces additional cognitive control via pre-frontal cortical (PFC) regions following the detection of conflict by the anterior cingulate cortex (ACC). In stimulus-response compatibility (SRC) tasks, conflict can be expected in incongruent trials, in which one stimulus attribute signals the opposite response to the target response. Selection of the target response suffers interference by activation of the opposite response, leading to delayed responses and more errors. The current study used Event Related Potentials (ERPs) to analyze two forms of interference in SRC tasks, and the means by which cognitive control helps to resolve such interference.

2.1.1 Stimulus-Response Compatibility and Event Related Potentials

One method of analyzing the temporal dynamics of interference and its resolution is by means of the Lateralized Readiness Potential (LRP). The readiness potential is the negative-going activation recorded over the motor cortex contralateral to a hand movement, which gradually increases up until response execution (Kornhober & Deecke, 1965). The LRP is a difference wave, which is calculated by subtracting the activation recorded ipsilateral to the response hand from the activation recorded contralateral to the response hand, and then averaging over left and right-hand responses (Coles, 1989; de Jong, Wierda, Mulder, & Mulder, 1988; Gratton et al., 1988). When calculated according to this method, the resulting difference wave represents correct response activation as negative deflections and incorrect response activation as positive deflections. LRPs are particularly relevant to an analysis of interference in SRC tasks due to the finding of a deflection of incorrect-response activation prior to the correct-response activation in incongruent trials (Gratton et al., 1988). This deflection has been referred to as the “Gratton dip”, and has been interpreted as motor-related activation of the incorrect response (Coles, 1989). Interestingly, the size, timing, and topography of the dip in lateralized ERPs (L-ERPs) is dependent upon many factors, as will be discussed below.

The most frequently investigated electrophysiological correlate of cognitive control is the latency and amplitude of N2, a negative ERP component peaking 200 ms or later following stimulus onset. N2 is assumed to reflect the selection of the appropriate response (Gajewski, Stoerig, & Falkenstein, 2008; Gajewski, Kleinsorge, & Falkenstein, 2010), the inhibition of inappropriate responses (Carriero, Zalla, Budai, & Battaglini, 2007; Falkenstein, Hoormann, & Hohnsbein, 1999; Falkenstein, Hoormann & Hohnsbein, 2002; Heil, Osman, Wiegelmann, Rolke, & Henninghausen, 2000; Kopp, Rist, & Mattler, 1996; van Boxtel, van der Molen, Jennings, & Brunia, 2001) or conflict processing (Carter & van Veen, 2007; Nieuwenhuis, Yeung, van den Wildenberg, & Ridderinkhof, 2003; Donkers & van Boxtel, 2004; van Veen & Carter, 2002; Yeung et al., 2004). When interference in incongruent trials is resolved, leading to a correct response, N2 is often enhanced and/or delayed relative to correct congruent trials (Bartholow, Pearson, Dickter, Sher, Fabiani, & Gratton, 2005; Carriero, Zalla, Budai, & Battaglini, 2007; Gajewski, Stoerig, & Falkenstein, 2008; Heil, Osman, Wiegelmann, Rolke, & Henninghausen, 2000; Kopp, Rist, & Mattler, 1996; Melara, Wang, Vu, & Proctor, 2008; Purmann, Badde, Rodriguez, & Wendt, 2011; van Veen & Carter, 2002; Wendt, Heldmann, Münte, & Kluwe, 2007). Common to all of these hypotheses, cognitive control is needed to resolve interference, and N2 is assumed to reflect cognitive control (for a review, see Folstein & van Petten, 2008).

2.1.2 Interference Effects in Eriksen and Simon Tasks

This study compares two well-used SRC tasks, both of which can be used to demonstrate visual spatial interference effects: the Eriksen flanker task (Eriksen & Eriksen, 1974) and the Simon task (Simon & Rudell, 1967). Both tasks require deciding between a left- or a right-hand response. In the Eriksen flanker task, a central target stimulus (e.g. an arrow or a letter) is surrounded by noise (flanker) stimuli that are either the same or different to the target. Typically, responses are facilitated when the flankers signal the same response as the target, and hindered when the flankers signal the opposite response (Eriksen & Eriksen, 1974; Kopp et al., 1996; Ridderinkhof, van der Molen, & Bashore, 1995; van Veen & Carter, 2002; Wendt, Heldmann, Münte, & Kluwe, 2007). In the Simon task participants are asked to respond with a left- or right-hand response to a symbolic feature of the stimulus (e.g. shape or color). The stimulus is presented to the left or right side, implying that stimulus location can be spatially congruent or incongruent with the target response-hand. Relative to

centrally presented stimuli, performance is typically facilitated by congruent stimulus locations and hindered by incongruent locations (Hommel, 1993; Leuthold & Schroter, 2006; Masaki et al., 2007; Melara et al., 2008; Simon & Rudell, 1967; Wendt, Kluwe, & Peters, 2006).

Few studies have compared Eriksen and Simon effects. Two previous studies compared performance measures between the Eriksen and Simon tasks (Stoffels & van der Molen, 1988; Stins, Polderman, Boomsma, & de Geus, 2005), and both found that interference effects were larger for incongruent flankers than for incongruent stimulus locations. So far no studies have used electrophysiological measures to compare interference in the Eriksen and Simon tasks. Such a comparison might clarify the extent to which these similar forms of interference affect the same processes. However, we will now turn to the many studies that have used ERPs to investigate one of these tasks separately.

In the Simon task early asymmetries have been found to reflect more than just preferential response activation, as can be seen in Lateralized ERPs (L-ERPs). Response-related LRP deflections peak over central areas (usually C3'and C4') around response execution, and have been found to represent covert response activation (Leuthold & Jentzsch, 2002). However, with lateralized stimuli such as in Simon tasks, early deflections coincide with larger asymmetries at parietal areas, suggesting a carry-over effect from visual to motor areas (Praamstra & Oostenveld, 2003; Valle-Inclán, 1996; van der Lubbe, Jaśkowski, Wauschkuhn, & Verleger, 2001; Wascher & Wauschkuhn, 1996). These early parietal asymmetries with lateralized Simon stimuli peak at around 200 ms following stimulus onset. Extensive research with the Simon task has investigated the lateralization of N2 as well as its contribution to L-ERPs. Lateralized N2 at parietal areas is maximal contralateral to the stimulus location (N2pc) and is assumed to reflect spatial attention-related processes (Eimer, 1996; Luck & Hillyard, 1994; Praamstra and Oostenveld, 2003). Similarly, lateralized N2 at central areas is also maximal contralateral to the stimulus location (N2cc) and has been interpreted as reflecting spatial attentional-motor-related processes (Leuthold & Schröter, 2006; Praamstra and Oostenveld, 2003).

In the Eriksen task trials with incongruent flankers exhibit early activation of the incorrect response in LRPs from about 250 ms following stimulus onset (Gratton et al., 1988; Heil et al., 2000; Kopp et al., 1996; van 't Ent, 2002; Wascher et al., 1999; Willemssen et al., 2004). Wascher and collegues (1999) used a standard Eriksen task with flankers presented

above and below the target, and reported a dip at parietal areas with a similar timing to the dip at central areas. Willemssen and colleagues (2004) found the same dip at parietal areas using vertically-oriented arrows, excluding the possibility of attention-related asymmetries. Both studies referred to the dip at parietal areas as Direction Encoding Lateralization (DEL), and interpreted it as pre-motor encoding of response direction via a visuo-motor pathway in the brain. The DEL found in the Eriksen task is assumed to reflect primarily response-related processes, but N2pc and N2cc found in the Simon task are assumed to reflect primarily attention-related processes. A direct comparison of the Simon and Eriksen tasks with L-ERPs should reveal the extent or absence of this difference.

Interference by incongruent flankers has been found to enhance the amplitude of a fronto-central N2 peaking at around 300 ms post-stimulus (Bartholow et al., 2005; Heil et al., 2000; Kopp et al., 1996; Purmann et al., 2011; van 't Ent, 2002; van Veen & Carter, 2002; Wendt et al., 2007). In the Simon task, an enhanced N2 for incongruent trials has been reported with visual (e.g. Carrierro et al., 2007) and auditory stimulation (Melara et al., 2008). Collectively, previous studies suggest that it is safe to assume that fronto-central N2 reflects cognitive control (Folstein & van Petten, 2008). By comparing N2 effects in the Eriksen and Simon task, inferences can be made concerning the timing and sensitivity of the control processes involved in resolving each type of interference.

2.1.3 Hypotheses in Terms of a Dual-Process Model of Stimulus-Response Compatibility

ERP measures of interference and cognitive control appear to involve different timing and topography in brain potential analyses of the Eriksen task compared to brain potential analyses of the Simon task. Furthermore, the larger interference effects found in behavioral comparisons for the Eriksen task compared to the Simon task suggest that resolving interference requires more cognitive control in the Eriksen task than in the Simon task. The most frequently cited models of stimulus-response compatibility assume that interference is a result of dual-processes (e.g. de Jong, Liang, & Lauber, 1994; Kornblum, Hasbroucq & Osman, 1990; Ridderinkhof et al., 1995). The first process is direct *unconditional* perceptual-response activation by shared features between stimulus and response (dimensional overlap, c.f. Kornblum, Hasbroucq & Osman, 1990). The second process is indirect *conditional* selection of the relevant features and the appropriate response according to the currently active symbolic stimulus-response binding (e.g. left-pointing arrow = left hand

response). This implies that when deciding between a left or right hand response, spatial stimulus attributes will quickly activate a response via direct *unconditional* processes (S-R priming), and stimulus attributes that share the same symbolic codes as the response will activate the response more slowly via indirect *conditional* processes.

In terms of the tasks investigated here, we assume that the spatial asymmetry inherent in horizontal Simon stimuli is particularly effective at *unconditional* S-R priming and should therefore lead to the earliest deflections in L-ERPs. On the contrary, we expect that Eriksen interference reflects a disturbance to indirect *conditional* processes when selecting the target/response. In other words, in the Eriksen task we assume that *conditional* processes initially select the response associated with the distracter arrows (as well as the target arrow), due to their relevant symbolic stimulus-response codes. This hypothesis predicts that interference effects (visible in L-ERPs) will be later and should involve different brain topography in the Eriksen task than in the Simon task because activation of a response by flanker arrows will be dependent upon indirect conditional processing. Although the dual-process model does not specify the ERP topography of conditional and unconditional processes, it does allow clear predictions concerning the timing of interference and its resolution. We are interested in the timing of preferential response activation in each task, and specifically whether the peak of N2 is lateralized with both Simon and Eriksen interference. If preferential response activation is indirect (conditional) in the Eriksen task, then resolution of interference should also be later in the Eriksen task, predicting larger interference effects on RTs and later effects of cognitive control. Most interesting is whether the timing and topography of cognitive control (assessed as the amplitude and latency of N2) follows the same pattern as interference in L-ERPs. Therefore, we expect an early N2 enhancement for incongruent Simon stimuli, and a later N2 enhancement for incongruent flankers.

2.2 METHODS

2.2.1 Participants

Fifteen right-handed volunteers (7 women and 8 men), ages ranging from 20 to 31 (mean 24) years gave informed consent to participate in the study. Twelve of the participants were students who received course credits for their time, and three participants volunteered to

take part without receiving compensation. All reported to be neurologically healthy and had normal or corrected-to normal vision.

2.2.2 Apparatus

Participants were seated in a comfortable chair in a dimly lit, ventilated, soundproofed, electrically shielded room. Stimuli were displayed on a 14-inch monitor positioned 130 cm from the participant. Responses were measured and A/D converted on-line from Kyowa ML-20KA zero-displacement force transducers, positioned under the participant's index fingers in custom-made hand-supports. The transducers recorded the build-up of force, whereby only responses achieving at least 15 percent of the participant's maximum force were treated as valid reactions. Reaction times were calculated, for valid reactions only, as the moment at which the force reached two percent of the participant's maximum force.

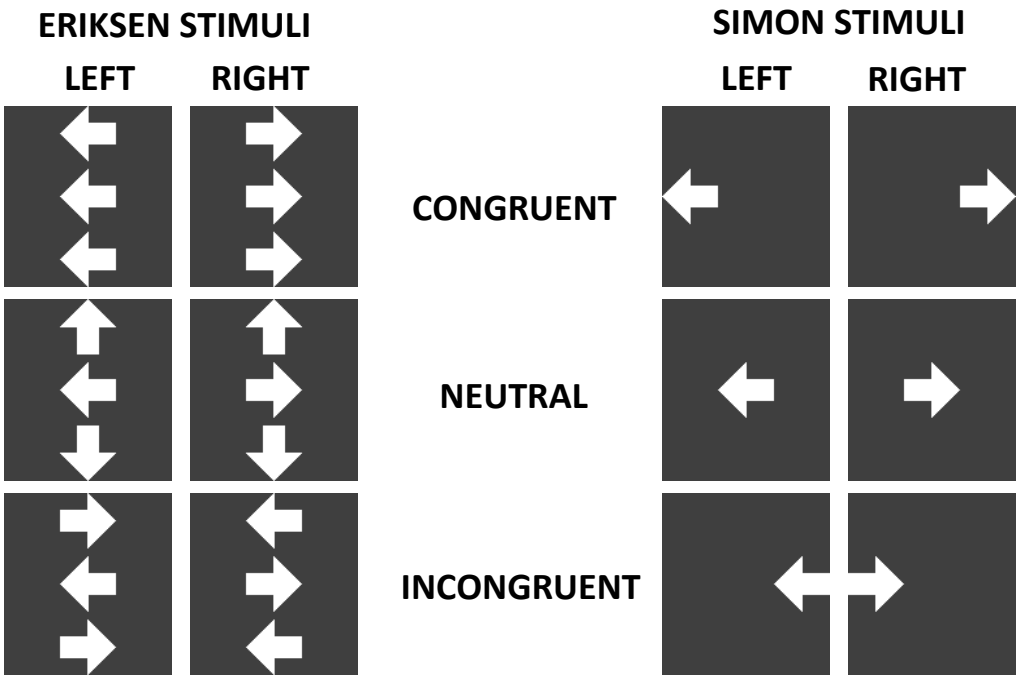

Figure 2.1. Detail of the stimuli used in the Eriksen task (left) and in the Simon Task (right).

2.2.3 Stimuli and Design

White arrow stimuli, each measuring 1.4 cm (0.62°) in width and height, were presented against a dark gray background (see Figure 2.1). In the Simon task one arrow, pointing either left or right, was presented either at fixation or displaced 1.3 cm (0.57°) to the left/right of fixation, such that the location of the arrow was congruent, neutral, or incongruent to its orientation. In the Eriksen task three arrows were presented in a vertical array (similar to

Wascher et al., 1999). The total array measured 4.6 cm (2.0°) in height, consisting of one target arrow at fixation, flanked by one arrow above and one arrow below. The target arrow pointed either left or right, and the flanker arrows pointed either in the same direction as the target (congruent), in the opposite direction (incongruent), or away from the target (neutral). Arrows instead of another shape were used as neutral flankers in order to avoid pop-out effects. In both the Simon and the Eriksen flanker task, one third of the trials were neutral, one third congruent, one third incongruent, and half of the targets in each Congruency condition pointed left and half of them pointed right.

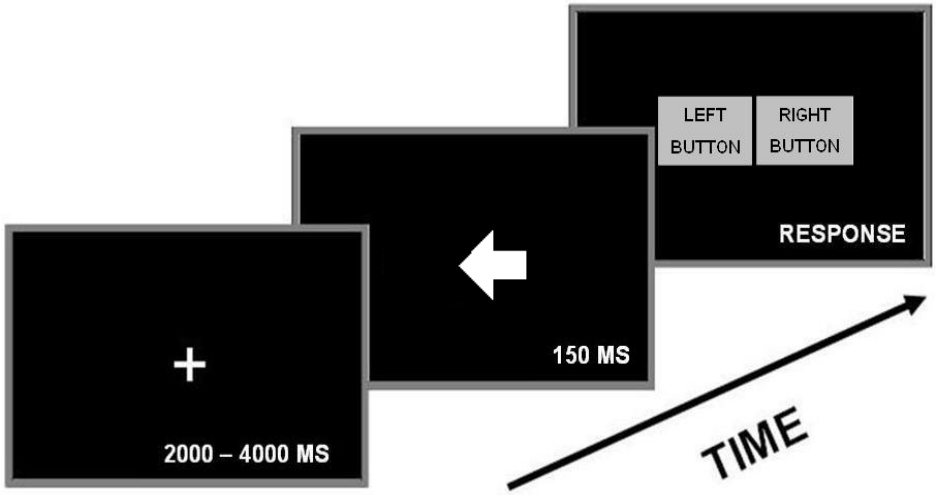

Figure 2.2. Example of a neutral trial in the Simon Task.

2.2.4 Procedure

Participants' maximum force on the force transducers was measured at least three times for each hand, the average of which was used to determine response validity. Only responses achieving at least 15 percent of the participant's maximum force were treated as valid reactions, and of these valid responses the reaction time was determined as the point at which the force achieved 2 percent of the participant's maximum force. All participants completed both tasks (Simon/Flanker), whereby task order was counter-balanced. Each task consisted of three test blocks (each comprising 180 trials), preceded by 2 short practice blocks (36 trials each). Test blocks incorporated 5 sub-lists of 36 randomized stimuli, such that each condition (congruency x response hand) occurred just as often in the beginning, end or middle of the complete list. To avoid excess artifact in the electrophysiological data, participants were asked to sit as still as possible, to keep their eyes on the fixation cross during each test block, and to avoid moving their eyes to the location of the displaced stimuli

during the Simon task (eye movements were recorded and trials with saccades were rejected prior to analysis, as described below). In the Simon task, participants were asked to give a spatially compatible response to the orientation of the arrow, ignoring its location. In the Eriksen flanker task, participants were asked to give a compatible response to the orientation of the central arrow, ignoring the flanker arrows. Both tasks involved responding as quickly as possible, by pressing the appropriate index finger briefly but firmly, without making too many errors. Performance feedback was given ("too weak", "wrong button", "multiple buttons", "too late", or "good") during the practice blocks, but not during test blocks. The trial began with the presentation of a fixation cross at the centre of the screen, replaced after 2000-4000 (random variable ITI) by the stimulus for 150 ms. The fixation cross remained on the screen until the participant responded or the deadline was reached (1200 ms), at which point the next trial was initiated (see Figure 2.2). Each test block lasted approximately 9.5 minutes, and was followed by a short break. The entire experimental session lasted approximately 3 hours.

2.2.5 Electrophysiological Recording

EEG was recorded from Cz, Fcz, Fz, Pz, C3, P3, F3, C4, P4, F4, M1 and M2 using Beckman Ag/AgCl cup electrodes with a diameter of 8 mm, affixed to the scalp with Grass EC-2 electrode paste. Horizontal EOG was recorded from the lateral canthus of each eye, and vertical EOG from above and below the right eye, using Beckman Ag/AgCl electrodes with a diameter of 2 mm, affixed with Signa gel. Impedance of all electrodes was kept to less than 5kΩ. Signals were amplified with a 0.053 Hz high-pass filter and a 30 Hz low-pass filter, and A/D converted at 200 Hz.

2.2.6 Data Analysis

Stimulus and response markers in the raw data were coded from the behavioral data, in order to exclude errors and outliers. All EEG channels were referenced to average mastoids and band-pass filtered from 0.1 to 12 Hz. The filtered data were segmented into epochs ranging from 100 ms prior until 800 ms following stimulus onset. Blinks (in 47% of segments) were detected and corrected in all EEG channels, using the regression technique (Gratton & Coles, 1983) incorporated in the Brain Vision Analyzer program (Brain Products GmbH). Segments with artifacts in EEG (3% of segments) were rejected using an automatic

procedure that rejected all channels of segments that had a voltage step larger than 100 μV. Horizontal eye movements were calculated (right-left), low-pass filtered at 5 Hz, and segments containing saccades (horizontal EOG with an amplitude greater than +/- 75 μV) were removed automatically (7% of segments) in order to exclude differences in eye-movement between tasks. Segments were baseline corrected to the 100 ms prior to stimulus presentation, and stimulus-locked averages were calculated for each subject according to condition (based on a minimum of 28 segments per subject per condition).

For visual comparison purposes, L-ERPs were calculated according to the LRP formula: [C4(left-response)-C3(left-response)+C3(right-response)-C4(right-response)] / 2. Correct response activation was therefore represented by negative amplitudes. In order to assess lateralized components statistically, analyses were performed on contra/ipsi-lateral waveforms (c.f. Yordanova, Kolev, Hohsbein, & Falkenstein, 2004). Waveforms were calculated separately for electrodes contralateral and ipsilateral to the direction of the target arrow (*response hand)*, for F3/4, C3/4 and P3/4. The mean amplitude was exported for each condition in two separate intervals, corresponding to the timing of components assumed to represent S-R Priming (160-190 ms) and N2cc (270–300 ms). In order to assess cognitive control, midline components were scored as the mean amplitude in a 30 ms interval around the peak of the components in the grand averaged ERPs; N2 (270–300 ms) and N350 (340–370 ms). The within-subjects factors included in the multivariate analyses of variance for lateralized components were: Task, Congruency, Topography (frontal/central/parietal), and Lateralization (contralateral/ipsilateral). Analyses of midline components included electrode FCz as well as Fz, Cz, and Pz, involving the factors: Task, Congruency, and Electrode.

2.3 RESULTS

2.3.1 Behavioral Results

Initial behavioral analyses revealed no main or interaction effects of Response Hand, and the data associated with each hand were collapsed for the remaining analyses, involving the factors Task (Eriksen/Simon) and Congruency (Congruent, Neutral, Incongruent). The data are summarized in Figure 2.3. Overall mean RT was 374 ms, with 5 percent errors. More errors were made in incongruent trials (10%) than in congruent trials (1%), $F(2,13) = 22.2$, $p<.001$, $\eta_p^2 = .774$. Congruency effects on RT were larger in the Eriksen task (difference

incongruent - congruent = 60 ms) than in the Simon task (difference incongruent - congruent = 39 ms), supported by an interaction between Task and Congruency ($F(2,13) = 13.1$, $p = .001$, $\eta_p^2 = .668$). Separate RT analyses per task confirmed the Congruency effect in both tasks (Eriksen, $F(2,13) = 233.4$, $p<.001$, $\eta_p^2 = .973$; Simon, $F(2,13) = 108.2$, $p<.001$, $\eta_p^2 = .943$). All pair-wise Congruency comparisons of RTs were significant in the Eriksen task ($ts(14) > 16.1$, $ps<.001$) and in the Simon task ($ts(14) > 3.8$, $ps<.01$). Pair-wise comparisons between tasks revealed that both neutral and incongruent trials were slower in the Eriksen task than in the Simon task ($ts(14) > 2.9$, $ps<.05$). In sum, both tasks revealed interference effects, which were larger in the Eriksen than in the Simon task.

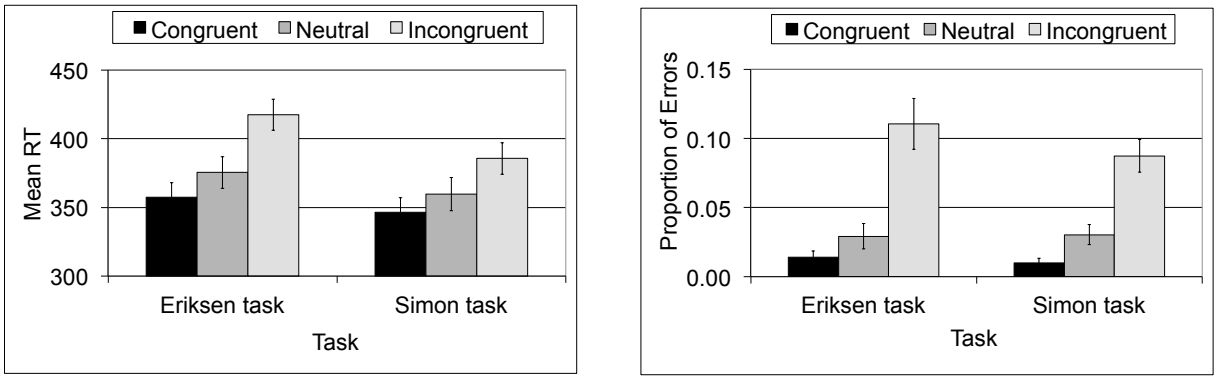

Figure 2.3. Overall mean RTs (left) and errors (right) in Eriksen and Simon tasks.

2.3.2 Preferential Response Activation

Stimulus-locked L-ERPs are depicted in Figure 2.4. Preferential activation of the incorrect response in incongruent trials had an early (175 ms) parietal maximum in the Simon task and a later (285 ms) central maximum in the Eriksen task. Stimulus-locked waveforms are displayed in the Eriksen task (Figure 2.5) and in the Simon Task (Figure 2.6), according to Congruency, Topography and Lateralization. Lateralized waveforms (contralateral vs. ipsilateral to the target arrow/response) were analyzed to assess the interval of interference in each task. The first interval is referred to as Early S-R Priming (mean amplitude between 160 – 190 ms), and the second interval is referred to as Lateralized N2 (mean amplitude between 270 – 300 ms).

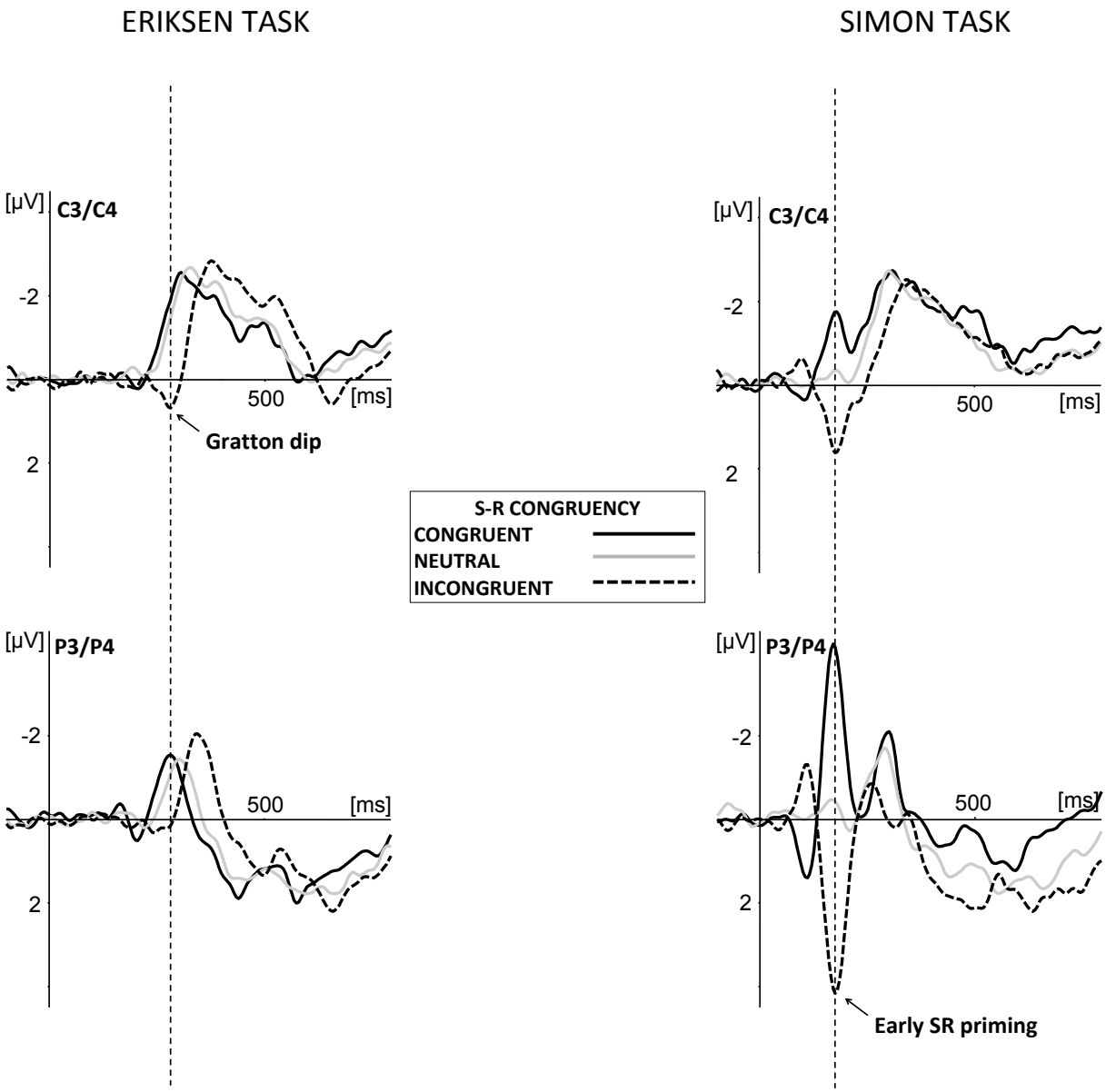

Figure 2.4. Stimulus-locked L-ERPs at C3/C4 (top) and P3/P4 (bottom), for the Eriksen task (left), and for the Simon task (right). L-ERPs are separated according to S-R Congruency, for congruent (black solid line), neutral (gray solid line), and incongruent (black dashed line) trials. The dashed line indicates the maximum of preferential response activation in each task.

2.3.2.1 Early S-R Priming

As expected, only in the Simon task were asymmetries visible in this early interval in lateralized ERPs (compare Figures 2.4, 2.5 and 2.6). Initial analyses of this interval resulted in a four-way interaction ($F(4,11) = 22.2$, $p<.001$, $\eta_p^2 = .890$). The asymmetry visible in LRPs for the Simon task (Figure 2.4, right panel) reflected an enhancement to an early positive component at parietal electrodes in the Simon task (Figure 2.6), contralateral to the

lateralized stimulus, and was supported by an interaction at parietal electrodes in only the Simon task between Congruency and Lateralization ($F(2,13) = 67.3$, $p<.001$, $\eta_p^2 = .912$). Also in the Simon task only, an additional early positive component appeared at central electrodes ipsilateral to the stimulus in the same interval (Congruency x Lateralization interaction at central electrodes in only the Simon task, $F(2,13) = 58.0$, $p<.001$, $\eta_p^2 = .899$). In order to assess statistically the parietal maximum for Congruency effects in the Simon task (see figure 2.6, contralateral electrodes), early S-R Priming was analyzed in the Simon task at contralateral electrodes, which supported the parietal maximum (Topography x Congruency interaction, $F(4,11) = 16.4$, $p<.001$, $\eta_p^2 = .857$).

2.3.2.2 Lateralized N2

The lateralization of N2 at central electrodes was influenced by Congruency specifically in the Eriksen task, resulting initially in a four-way interaction ($F(4,11) = 12.2$, $p<.001$, $\eta_p^2 = .817$), and a three-way interaction at central electrodes ($F(2,13) = 8.5$, $p = .004$, $\eta_p^2 = .567$). At central electrodes contralateral to the response (see Figure 2.8), N2 was reduced specifically by incongruent flankers (Task x Congruency interaction, $F(2,13) = 5.7$, $p = .017$, $\eta_p^2 = .468$). Analyses were then performed separately according to Task. At central electrodes in the Eriksen task, N2 had a contralateral (to the response) maximum in congruent trials and an ipsilateral maximum in incongruent trials (Congruency x Lateralization interaction, $F(2,13) = 35.8$, $p<.001$, $\eta_p^2 = .846$). In contrast, in the Simon task N2 had a contralateral maximum in *all* congruency conditions, $F(1,14) = 25.6$, $p<.001$, $\eta_p^2 = .647$. In order to assess statistically the central maximum for Congruency effects in the Eriksen task (see figure 2.5, contralateral electrodes), lateralized N2 was analyzed separately in the Eriksen task at contralateral electrodes, which supported the central maximum (Topography x Congruency interaction, $F(4,11) = 10.6$, $p = .001$, $\eta_p^2 = .794$).

In sum, preferential response activation in the Simon task was early and had a parietal maximum. In contrast, preferential response activation with incongruent flankers in the Eriksen task was later, reflected by a lateralized N2, and had a central maximum. Interestingly, Lateralized N2 was sensitive to Congruency only in the Eriksen task, but not in the Simon task.

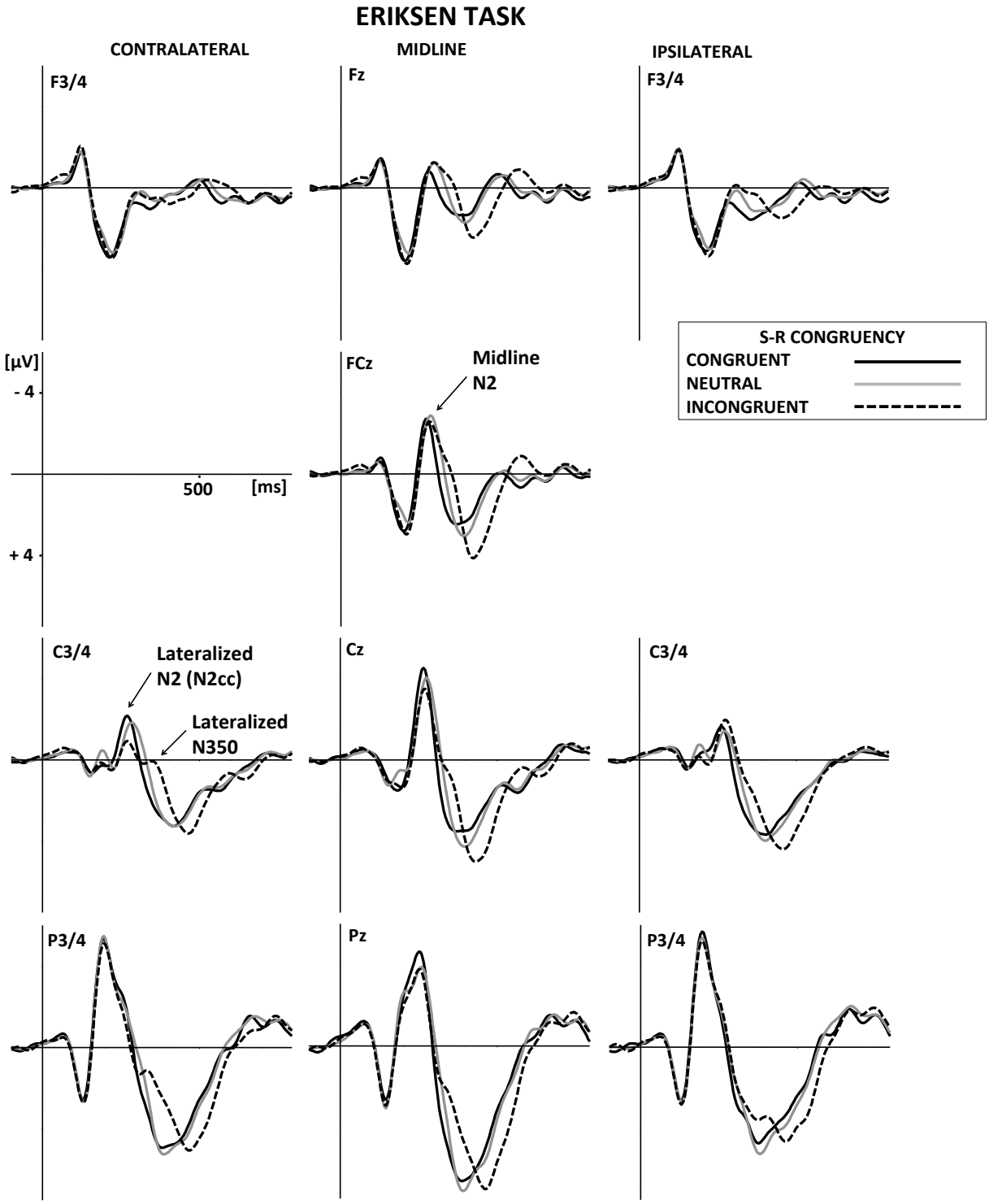

Figure 2.5. Stimulus-locked ERPs in the Eriksen task, separated for waveforms at midline, and contra- vs. Ipsilateral to the response hand (arrow direction).

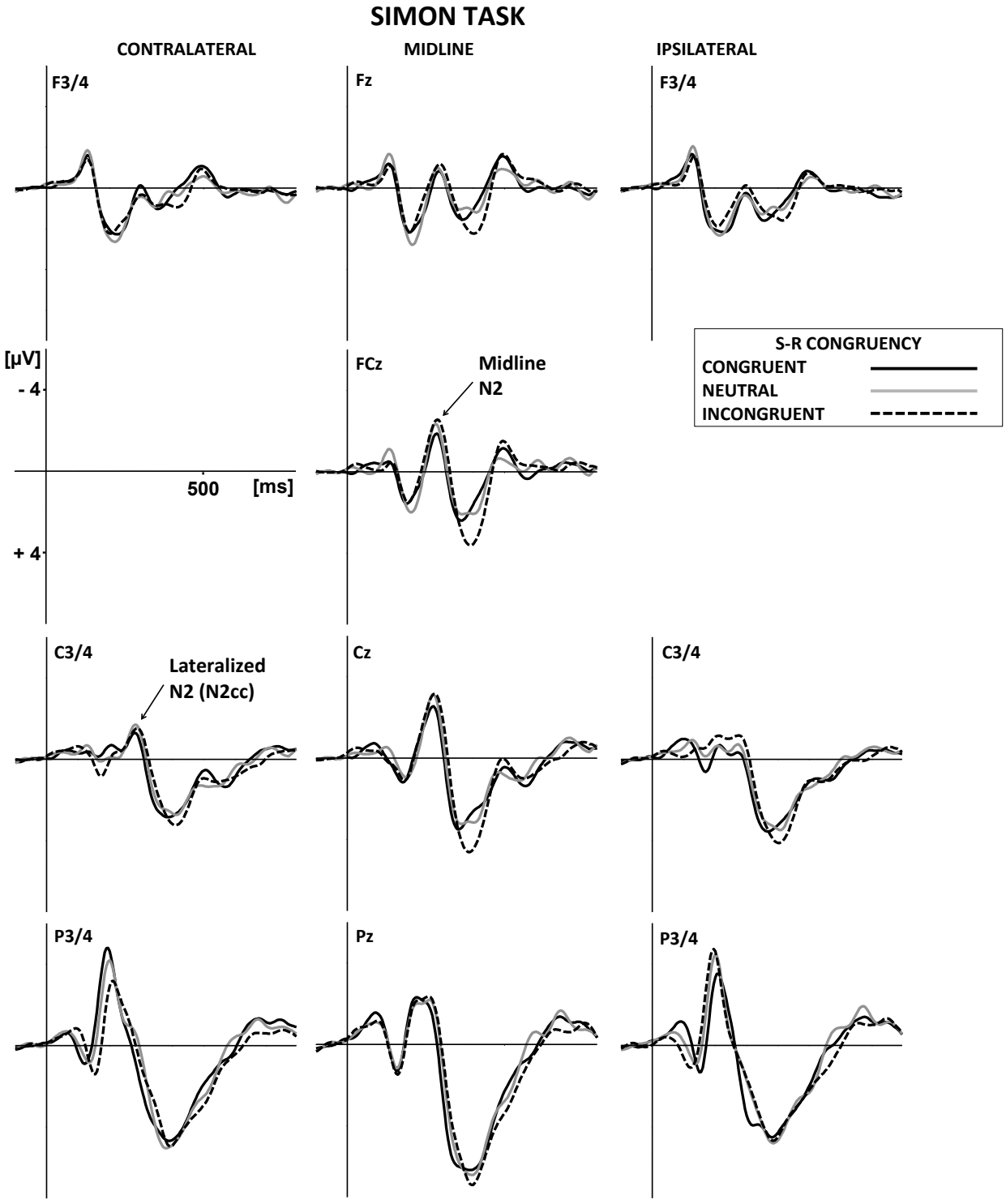

Figure 2.6. Stimulus-locked ERPs in the Simon task, separated for waveforms at midline, and contra- vs. Ipsilateral to the response hand (arrow direction).

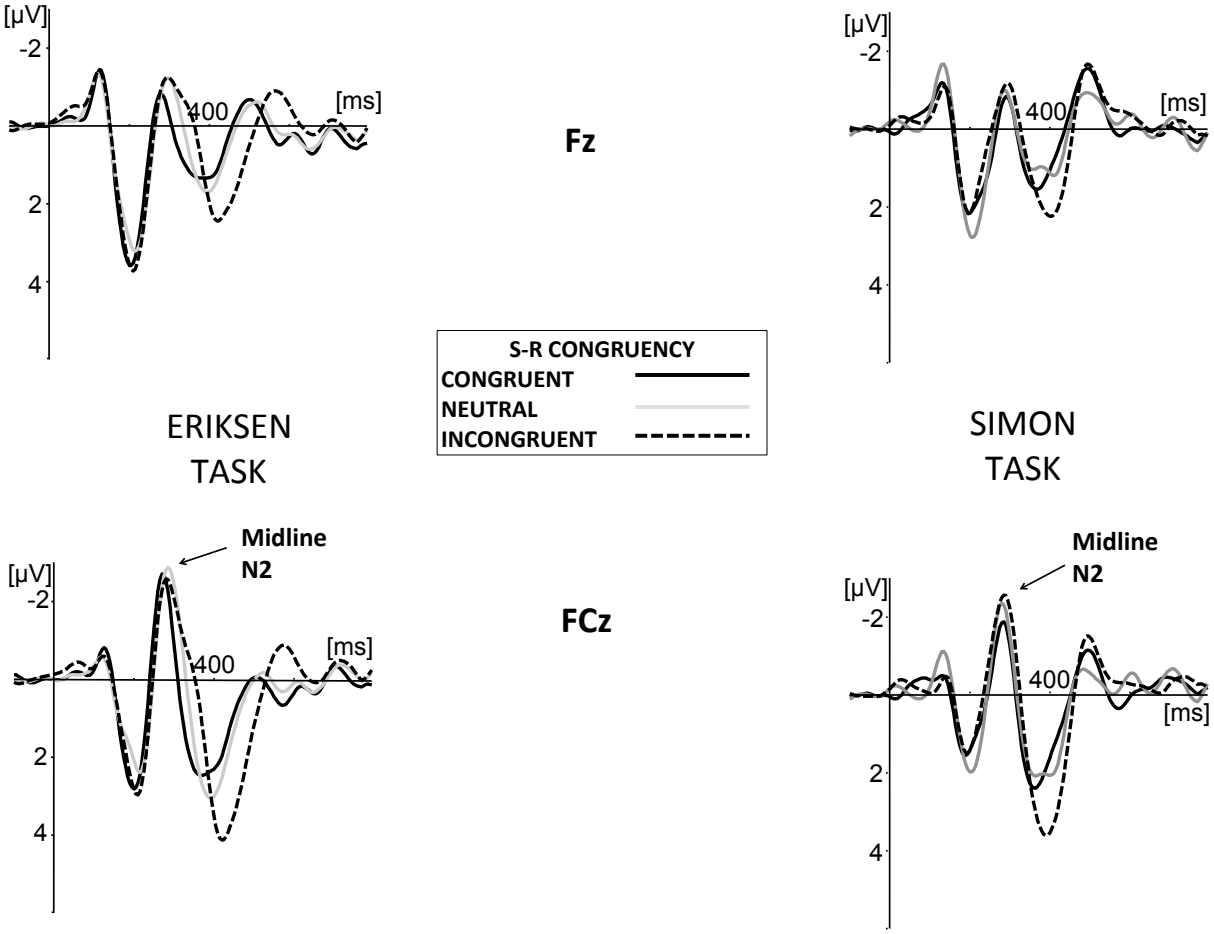

Figure 2.7. Stimulus-locked midline ERPs at Fz (top) and FCz (bottom) , for the Eriksen task (left) and the Simon task (right). ERPs are separated according to S-R Congruency, for congruent (black solid line), neutral (gray solid line), and incongruent (black dashed line) trials.

2.3.3 Cognitive Control

2.3.3.1 Midline N2

Midline N2 was analyzed as the mean amplitude in the same interval used for analysis of Lateralized N2. Midline ERPs at Fz and FCz are presented in Figure 2.7 for each task. Midline N2 was maximal with incongruent stimulus locations in the Simon task, but with neutral flankers in the Eriksen task (interaction between Task & Congruency, $F(2,13) = 4.7$, $p = .030$, $\eta_p^2 = .418$). Separate analysis of the Simon task revealed that although the Congruency pattern was in the expected direction, the effect failed to attain acceptable significance ($F(2,13) = 3.1$, $p = .081$, $\eta_p^2 = .321$). Separate analysis of the Eriksen task revealed that the Congruency pattern differed according to electrode (interaction between Congruency & Electrode: $F(6,9) = 4.2$, $p = .027$, $\eta_p^2 = .736$), but none of the Congruency patterns attained significance in separate analyses per electrode. T-tests within tasks revealed only that N2

was enhanced for neutral flankers relative to incongruent flankers at Cz, $t(14) = 2.63$, $p = .020$. T-tests between tasks revealed that N2 was larger in the Eriksen task than in the Simon task at Cz in neutral trials, and at Pz in both congruent and neutral trials, all $ts(14) > 2.4$, $ps < .03$. In sum, midline N2 was most sensitive to incongruent locations and neutral flankers.

2.3.3.2 N350

In line with the expectation of later cognitive control with flanker interference, N2 was visibly wider for incongruent trials in the Eriksen task (see figure 2.5, particularly Fz), suggesting an additional negative component at around 350 ms. Initial analyses of midline N350 (mean amplitude 340-370 ms) resulted in a three-way interaction between Electrode, Task and Congruency ($F(6,9) = 5.8$, $p = .01$, $\eta_p^2 = .795$), and subsequently separate analyses were conducted per Task. As expected, enhancement to N350 for incongruent trials was unique to the Eriksen Task. In the Eriksen task, the enhancement to N350 for incongruent flankers varied per electrode (interaction between Congruency and Electrode, $F(6,9) = 8.4$, $p = .003$, $\eta_p^2 = .849$), with the largest congruency effects at FCz ($F(2,13) = 6.9$, $p = .009$, $\eta_p^2 = .513$). In the Simon task, only the effect of Electrode attained significance ($F(3,12) = 11.1$, $p = .001$, $\eta_p^2 = .735$), such that frontal electrodes were associated with the most negative values and Pz with the most positive values. In sum, specifically the interference in incongruent flanker trials resulted in late effects at N350.

Inspection of Figure 2.5 revealed that midline N350 for incongruent flankers was simultaneous to a component at central electrodes contralateral to the response, which was also specific to incongruent flanker trials (see Figure 2.8). Post hoc analyses of lateralized N350 resulted in a four way interaction between Topography, Lateralization, Task, and Congruency ($F(4,11) = 4.4$, $p = .023$, $\eta_p^2 = .616$), in which N350 was indeed most enhanced in incongruent flanker trials at central electrodes contralateral to the response. This contralateral location and incongruent flanker-related maximum was also confirmed by analyses at central areas (interaction between Task, Congruency, & Lateralization, $F(2,13) = 6.9$, $p = .009$, $\eta_p^2 = .514$). Separate analyses per Task at central areas revealed that the enhancement to N350 for incongruent trials was indeed significant in only the Eriksen task ($F(2,13) = 6.9$, $p = .003$, $\eta_p^2 = .582$), although the contralateral maximum of the component ($F(1,14) = 6.9$, $p < .001$, $\eta_p^2 = .766$) was not substantially influenced by Congruency

(interaction ns). In sum, lateralized N350 was most enhanced at central contralteral electrodes in incongruent flanker trials.

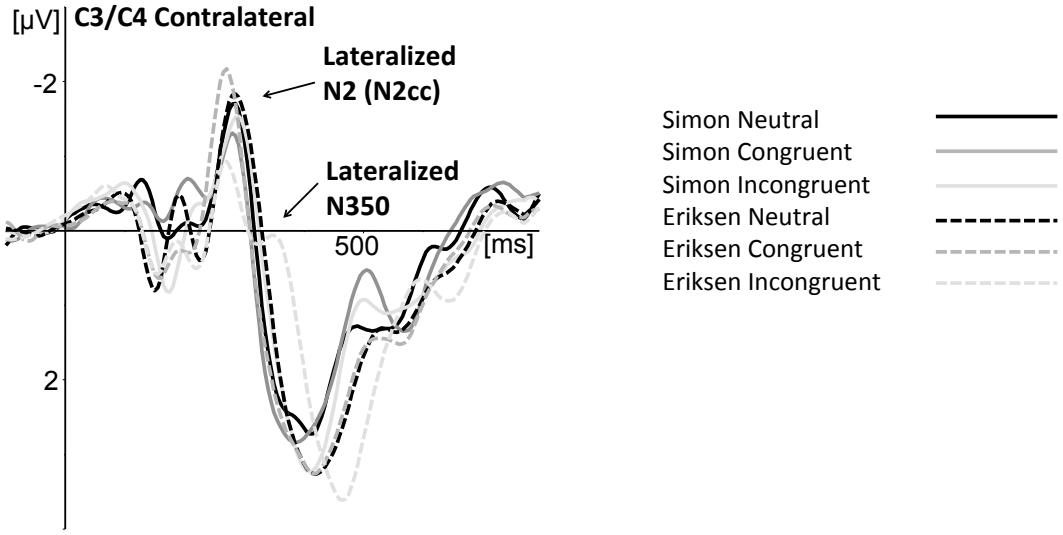

Figure 2.8. Stimulus-locked ERPs at C3/C4 contralateral to the response (N2cc), for the Simon task (solid lines) and the Eriksen task (dashed lines). ERPs are displayed separately according to S-R Congruency, for congruent (black), neutral (dark gray), and incongruent (light gray) trials.

2.4 DISCUSSION

The aim of this study was to assess whether Simon and Eriksen interference is induced and resolved via different processes within the context of a dual-process model. We hypothesized that only Simon interference reflects direct *unconditional* S-R priming, and that Eriksen interference is incurred via indirect *conditional* processes. Therefore interference should be both *induced* and *resolved* later in the Eriksen task, implying that behavioral interference effects should be larger, and that interference effects in brain potential measures will be later and should have different brain topography in the Eriksen task than in the Simon task. We found differences in both the induction and resolution of interference in the Eriksen task compared to the Simon task, and these differences can be interpreted in terms of the dual-process model as well as in relation to recent neural models of cognitive control.

Firstly, we found that interference effects are *induced* later in the Eriksen task than in the Simon task, affecting two separate deflections of preferential response activation, each with its own unique timing and topography. An early deflection, maximal at parietal electrodes, was present in the Simon task only. More than one hundred milliseconds later,

the lateralization of N2, maximal at central electrodes, was sensitive to Congruency in the Eriksen task only. The more posterior topography of preferential response activation in the Simon task compared to the Eriksen task suggests that visual asymmetry influenced the early appearance of preferential response activation in the Simon task. Although the perceptual differences between the Simon and Eriksen tasks can account for most of the difference early on in brain potentials, we assume that especially the dissociation between the tasks on the lateralization of N2 reflects the different timing of interference in each task. Lateralized N2 *was not* influenced by congruency in the Simon task, implying that interference was no longer present around the peak of N2 in the Simon task. On the other hand, lateralized N2 *was* sensitive to congruency in the Eriksen task, implying that interference was still present around the peak of N2 in the Eriksen task.

Secondly, we found that interference effects are *resolved* later in the Eriksen task than in the Simon task. Behavioral congruency effects were larger in the Eriksen task than in the Simon task. In brain potential measures, midline control components N2 and N350 were also differentially affected by the two tasks. Midline N2 was maximal with incongruent stimulus locations in the Simon task and with neutral flankers in the Eriksen task. This suggests that interference reflected at midline N2 was primarily perceptual, particularly considering that N2 was enhanced even more by neutral flankers than by incongruent stimulus locations. Midline N350 was enhanced uniquely by incongruent flankers, and lateralized N350 revealed that this late component was also maximal contralateral to the response.

The dissociation in both timing and topography suggest that interference affects different processes in each task. Specifically the finding that Simon interference had an effect on L-ERPs only prior to the peak of N2 suggests that Simon interference is resolved around the timing of the peak of N2. In terms of a dual-process model, we assume that Simon interference is incurred primarily via direct *unconditional* processes, independent of the specific stimulus-response binding. On the other hand, Eriksen interference probably reflects a disturbance to indirect *conditional* processes, whereby the flankers are initially processed according to the same stimulus-response binding as the target arrow. We assume that in the Simon task the direct *unconditional* nature of interference by stimulus location allowed this interference to be quickly resolved by cognitive control processes, around the

timing of N2. This interpretation relies on the finding that lateralized N2 in the Simon task was maximal contralateral to the response in all congruency conditions. On the other hand, with incongruent flankers, lateralized N2 was maximal ipsilateral to the response. Only around the timing of N350 was activation maximal contralateral to the response in all conditions, and particularly with incongruent flankers. In other words, in incongruent conditions interference was resolved around the timing of N2 in the Simon task, but interference was not resolved until the timing of N350 in the Eriksen task.

Particularly due to the limitations of the relationship between the processes in the dual-process model and brain topography, it is relevant to compare the current findings with recent models of conflict-monitoring and cognitive control (Carter & van Veen, 2007; Botvinick et al., 2001; Yeung et al., 2004). Although these models depend upon simulations for assessing the amount of conflict that leads to cognitive control, theoretically it is possible to compare preferential response activation to the concept of 'conflict'. According to conflict-monitoring models, conflict refers to competition between activated responses, implying that conflict will be greatest when two or more responses are equally active. Conflict-monitoring models assume that increased conflict leads to increased cognitive control, reflected in correct trials as an enhanced fronto-central N2. If the enhanced cognitive control components for incongruent trials in the current study reflect conflict, then we can deduct that the later cognitive control effects found in the Eriksen task (N350) imply that conflict was detected and/or resolved later in the Eriksen task than in the Simon task.

The lateralization of N350 in incongruent flanker trials also extends upon one finding of Praamstra and Oostenveld (2003). In their horizontal Simon task, even with incongruent stimulus locations lateralized N2 was maximal contralateral to the *stimulus* location. This implies that interference was not yet resolved by the peak of N2cc because response activation still favored the incorrect response. In the current Simon task, lateralized N2 was always maximal contralateral to the *response* location. We expect that this discrepancy between the studies reflects stimulus differences that influenced the amount of asymmetry, which affected the timing of parietal lateralizations. The study by Praamstra and Oostenveld involved bilateral presentation, in which the target stimulus was presented on one side of fixation and a filler on the other side. Although the intention of the authors was to minimize parietal lateralizations, such a stimulus array involved selection of the target over the filler.

On comparison of the L-ERPs in each study we expect that the reduced asymmetry inherent in their stimuli accounts for why early parietal lateralizations are smaller and peak earlier in their study (peaking around 155 ms) than in the current study (peaking around 175 ms). In their Simon task N2 also peaked earlier (245 ms) than in the current Simon task (285 ms), and N2 in their study coincided with interference at N2pc and N2cc. We expect that the complexity of their stimuli (bilateral presentation and letter targets rather than arrows), involving more selective processing, accounts for the later interference effects (at N2cc) in their Simon task compared to the current Simon task. It is likely that the additional selection processes involved in their design induced interference via *conditional* processing, reflected at N2cc.

Praamstra and Oostenveld (2003) performed source localization as well as functional analyses, locating N2cc to the lateral premotor cortex and interpreting it as being involved in the selection and inhibition of competing responses. In the current study we interpret the contralateral maximum at lateralized N2 in all Simon conditions to reflect activation of the appropriate response. However, with incongruent flankers the activation at lateralized N2 still favors the inappropriate response, implying that selection of the appropriate response in incongruent flanker trials takes place later than the peak of N2. But we assume that the control processes underlying N350 can account for the late selection of the appropriate response in incongruent flanker trials.

For a complete account of why the Eriksen and Simon tasks are assumed to suffer interference via different processes in a dual-process model, it is important to consider the different nature of the interference in each task. In the Eriksen task, both target and flankers can activate a response according to the same active symbolic codes/bindings. In the traditional Simon task, the target stimulus attribute is symbolic but the incongruent stimulus location shares spatial rather than symbolic response codes. In the current study, we interpret the brain potential data as support for interference by *unconditional* processes in the traditional Simon task, and by *conditional* processes in the traditional Eriksen task. However, depending on the task and stimuli, it is fair to assume that variations on the traditional Simon task could lead to more interference during *conditional* processes, such as when additional selection is necessary, involving attentional processes (e.g. the study by Praamstra & Oostenveld, 2003). Similarly, variations on the Eriksen task could lead to more

interference via *unconditional* processes, such as with larger stimulus arrays that have an inherent visual asymmetry.

An additional factor is also known to influence the extent of the interference effect in the Simon task, namely temporal overlap between S-R priming and response selection (Hommel, 1993, Wascher, 2005). We expect that the modest sensitivity of midline N2 to incongruent locations partially reflects the lack of temporal overlap between *unconditional* processing of the stimulus location (early S-R priming, around 175 ms) and the *conditional* selection of arrow direction (lateralized N2, around 285ms). Automatic S-R priming by spatial stimuli has previously been seen to appear and subsequently decay in LRPs if a response is not immediately executed (e.g. Eimer, 1995; Eimer, Hommel, & Prinz, 1995), implying that spatial S-R priming will only create interference if automatic response activation is still present during conditional response activation. In L-ERPs for the current Simon task, early S-R priming is seen to decay almost completely prior to final motor activation, and interference effects are not present at lateralized N2. We therefore assume that Simon effects in RTs and at N2 would have been larger if S-R priming had occurred later, allowing more temporal overlap with conditional response activation.

Many variations on the Simon task could lead to Simon effects being partially incurred via conditional processing. We assume that such interference is both *conditional* and *automatic*. Although traditionally *unconditional* processes were assumed to be automatic and *conditional* processes were assumed to be controlled, there is evidence that *conditional* processes are also automatic in S-R compatibility tasks (de Jong et al., 1994). Similarly, we assume that in the Eriksen task the flankers are automatically selected by conditional processes together with the central target. In fact, the finding of incorrect response activation by incongruent flankers suggests that initially response activation is influenced even more by the flankers than by the central target. It is likely that participants are often unable to focus on the central target prior to the onset of covert response activation. However, previously behavioral data have demonstrated that presenting Eriksen trials in a blocked design reduces the interference effect (Eriksen & Eriksen, 1974; Stoffels & van der Molen, 1988), presumably assisting perceptual selection. When selection of the target is effective, then selection of the response should require less effort. But if target selection is disturbed, more than one response will become active and response selection

will require increased control. This interpretation is supported by the combined findings in the Eriksen task of interference at the peak of lateralized N2 and a late enhancement at N350.

The incorrect response activation at parietal areas in L-ERPs with incongruent flankers is comparable to the Direction Encoding Lateralization (DEL) reported by Wascher et al. (1999) and Willemssen et al. (2004), which is assumed to reflect premotor response activation via a visuo-motor pathway. Just as with their studies, in the current experiment asymmetries in L-ERPs induced by incongruent flankers were smaller at parietal electrodes compared to central electrodes. Therefore, the topography and timing of the dip with incongruent flankers shed doubt on the possibility that it reflects the same visuo-spatial processes inherent to the visual Simon task. It is possible to differentiate between the DEL in the Eriksen task and the attention-related pathway that underlies S-R priming in the horizontal Simon task. Firstly the DEL coincides with the larger deflection at central areas, and secondly DEL has been reported in an Eriksen task with vertically-oriented arrows and responses (Willemssen et al., 2005) as well as with horizontally-oriented arrows and responses (Wascher et al., 1999). On the other hand, comparisons of the Simon effect in the horizontal and vertical dimension demonstrate that in the vertical dimension the LRP dip in incongruent trials is absent (Vallesi et al., 2005), and Simon interference is induced much more slowly (Wiegand & Wascher, 2005). These findings also suggest that only lateralized stimuli, such as used in the horizontal Simon task, induce direct spatial S-R priming, and other interference effects are incurred via cognitive coding (Wiegand & Wascher, 2005) or translation (Vallesi et al., 2005).

Similar to the studies by Wiegand and Wascher (2005) and Vallesi and colleagues (2005), the current findings suggest that the processes that underlie interference effects in the horizontal Simon task are unique to that task. We believe that both the current interpretation and the fundamental assumption by Wiegand and Wascher (2005) and Vallesi et al. (2005) are compatible with a dual-process account of stimulus-response compatibility. Such an account assumes that only lateralized or sufficiently asymmetrical stimuli can induce interference effects via direct *unconditional* processes, and other stimuli (including arrows) induce interference effects by activating the symbolic response codes via indirect *conditional* processes. Although interference can be induced *automatically* via both *unconditional* and

conditional processes (c.f. de Jong et al., 1994), we suspect that only unconditional response activation disappears automatically, therefore requiring little cognitive control if direct response activation is already decayed prior to conditional response activation. In contrast, we expect that conditional response activation does not decay automatically, but instead requires cognitive control (such as measured at N350) before it can be overcome.

In sum, we found support for the hypothesis that interference effects involve different processes in the traditional Simon task compared to the Eriksen flanker task. Only the lateralized stimuli in the Simon task induce fast spatial S-R priming. The incongruent flankers in the Eriksen task induce interference via slower indirect processes, which incur larger behavioral costs and require later cognitive control. Interestingly, we found that lateralized N2 is sensitive to interference in the Eriksen task, but not to interference in the traditional Simon task. We believe that the dissociation between the tasks in the timing of interference and cognitive control can be related to the dual-process model. Future variations on the traditional Simon and Eriksen tasks should aim to better differentiate between the effects of conditional and unconditional response activation in terms of brain topography.

Chapter 3:
Domain-specificity of conflict and control with Eriksen and Simon interference

This study used event related potential (ERP) measures to assess whether Eriksen (flankers) and Simon (locations) interference represent different mechanisms of conflict and control. We compared Simon and Eriksen interference within experimental blocks, and compatible and incompatible SR-mappings (tasks) between blocks. In line with a dual-route model, we assumed that Simon vs. Eriksen interference reflect direct- vs. indirect-route response activation, respectively, such that only Simon interference can be resolved by suppression of the direct route, whereas Eriksen interference should benefit from increased control via the indirect route. In compatible tasks, Simon effects were smaller than Eriksen effects in RTs, replicating previous results. In incompatible tasks, Eriksen effects were reduced and Simon effects reversed in relation to the target arrow direction, i.e. locations that were incongruent with the target arrow facilitated the incompatible response. In ERPs recorded over motor cortices, high-conflict conditions were associated with reduced negativity in a late N2 contralateral to the response: for Eriksen interference only in compatible tasks, and for Simon interference in both compatible and incompatible tasks, mirroring performance results. Midline N1 and N2 were enhanced for all Simon compared to Eriksen trials, implying that control (reflected at N2) was not triggered by response conflict but potentially by the detection of specific stimulus features (reflected at N1). We suspect that with mixed sources of conflict, Simon interference was mediated by reactive control suppressing the direct route, triggered by stimulus attributes. Eriksen interference is likely mediated by proactive control over task-bindings (indirect route effects), but in incompatible tasks direct- and indirect-route flanker interference might to some extent cancel each other out.

3.1 INTRODUCTION

Current models of cognitive control assume a feedback loop such that detection of response conflict via anterior cingulate cortex (ACC) triggers adjustments to cognitive control via prefrontal cortex (PFC) (Botvinick et al., 2001). While much work is underway to assess the neural mechanisms that support the conflict monitoring theory (for a review see Botvinick et al, 2004; Carter & van Veen, 2007), relatively little is known about the flexibility of cognitive control in its means for dealing with different forms of conflict. Tobias Egner (2008) has suggested that the limited evidence in favor of domain-specific cognitive control reflects methodological problems with studies that have attempted to identify independent control mechanisms. As Egner argued, in order to test the domain-specificity of cognitive control, each type of interference should reflect a different source of conflict. In a recent study (Mansfield et al., 2013) we found some support for domain-specific conflict resolution. However, that study used a blocked design, hence the results might have been influenced by different preparatory strategies between experimental blocks. In the current study, we present Simon and Eriksen interference in the same experimental blocks, and use performance and ERP measures to assess the domain-specificity of cognitive control involved in resolving each type of response conflict.

In the Eriksen task (Eriksen & Eriksen, 1974) response interference is manipulated using noise (flanker) stimuli that are either *congruent* (e.g. <<<<<) or *incongruent* (e.g. >><>>) with a central target. Typically, responses are faster and more accurate with *congruent* compared to *incongruent* flankers (Eriksen & Eriksen, 1974; Kopp et al., 1996; Ridderinkhof et al., 1995; van Veen & Carter, 2002; Wendt et al., 2007). Eriksen interference is traditionally accounted for by a continuous flow model, as opposed to a discrete stage model, such that simultaneous processing of both the central target and the flankers result in competing activation at the response level (e.g. Coles et al., 1985; Eriksen & Schultz, 1979; Eriksen et al., 1985; Smid et al., 1990). In fact, Eriksen interference probably reflects a combination of interference at both stimulus and response levels, because congruent arrays present multiple identical stimuli (leading to fast identification), whereas incongruent arrays present a combination of different stimuli (delaying the identification process). Inclusion of neutral trials can help to disentangle such S-S and S-R interference effects (e.g. Wendt et al., 2007; Lamers & Roelofs, 2011), and can be included as a control condition.

In the Simon task (Simon & Rudell, 1967), the task-relevant stimulus feature is non-spatial (e.g. green stimulus = green response button) and the task-irrelevant stimulus feature is spatial (e.g. location). Responses are generally faster when stimulus and response are spatially corresponding compared to noncorresponding (e.g. Hommel, 1993; Leuthold & Schröter, 2006; Masaki et al., 2007; Melara et al., 2008; Simon & Rudell, 1967; Wendt et al., 2006), which is referred to as the *Simon effect* (see Lu & Proctor, 1995, for a review). Eriksen and Simon effects are both well-known examples of how a task-irrelevant stimulus attribute (noise stimuli / stimulus location) can lead to automatic activation of an incorrect response. However, we have suggested that the nature of response conflict differs between Simon and Eriksen interference (Mansfield et al., 2013), which can best be accounted for in line with a dual-route model (e.g. de Jong, et al., 1994; Kornblum et al., 1990; Ridderinkhof et al., 1995).

3.1.1 Alternate Routes in a Dual-Route Model

Although both Eriksen and Simon interference reflect competition between competing responses, such response conflict might be generated via different routes in a dual-route model. Response activation is assumed to take effect simultaneously via two routes: a fast, direct route, which automatically processes any stimulus attributes that have shared features (dimensional overlap) with the response attributes; and a slower, indirect route, which intentionally translates the task-relevant stimulus attribute into the appropriate response. While response conflict with irrelevant stimulus locations (Simon interference) reflects automatic response activation via the direct route, we propose that response conflict with incongruent flankers (Eriksen interference) reflects automatic response activation via the indirect route of the model (c.f. Mansfield et al., 2013). This is because intentional processing of the target via the slower, indirect route of the model could generalize to the task irrelevant flanker stimuli.

Egner (2008) refers to the Dimensional Overlap model (Kornblum et al., 1990) as a means for differentiating between alternative sources of conflict. Importantly, interference by a task-irrelevant stimulus dimension can reflect either dimensional overlap between the irrelevant (interference) and relevant (target) stimulus attributes (S-S interference), dimensional overlap between the irrelevant stimulus attribute and response attributes (S-R interference), or both. In the case of Eriksen interference with arrow targets and distractors,

the flanker arrows have almost complete dimensional overlap with the target arrow (only the distance from fixation is different), and both target and flanker arrows have some dimensional overlap with the response (<=left and >=right). In the case of a Simon task in which arrows are the relevant dimension and stimulus location is the irrelevant dimension, the left/right location of the stimulus will have some dimensional overlap with the target arrow, but much more dimensional overlap with the response. As such, Eriksen and Simon interference with arrow targets share both sources of conflict, but to very different degrees.

3.1.2 Confirming Independent Sources of Conflict with a Reversed SR-Mapping

The difference in the source of conflict for Eriksen and Simon effects can be tested by measuring the effect of reversing the stimulus-response (SR) mapping instruction, so that participants are required to give an incompatible response (e.g. a left-pointing arrow requires a right-hand response). Generally, responses are slower and accuracy is lower when participants are required to give an incompatible response compared to a compatible response (Fitts & Deininger, 1954), referred to as the "mapping effect". If Eriksen interference reflects automatic response activation via the indirect-route (contradicting traditional continuous flow models, e.g. Eriksen & Schultz, 1979), then reversing the SR-mapping for an arrow target (via the slow, indirect route of the model) implies that flanker arrows will be subject to the same reversal, such that incongruent flankers will consistently facilitate the *incorrect* response, maintaining the Eriksen effect with either mapping.

In contrast, reversing the SR-mapping should make Simon effects appear to reverse (but only in terms of congruency with the target arrow), because a stimulus location that is incongruent with the target arrow will facilitate the correct (*incompatible*) response via the direct route. Some have suggested that when participants have to decide between a left and right hand response, reversing the SR-mapping in a traditional Simon task (e.g., green stimulus = red response button) does generalize to processing of the task-irrelevant stimulus location (de Jong et al., 1994; Hedge & Marsh, 1975; Valle-Inclan, 1996). Such 'logical recoding' accounts have been proposed in response to studies reporting actual reversed Simon effects, whereby the stimulus location facilitates the contralateral response (de Jong et al., 1994; Hedge and Marsh, 1975; Hommel, 1995; Stoffels & van der Molen, 1988). However, more recent studies have demonstrated that reversed Simon effects depend upon

the inclusion of trials in which stimulus location is the task relevant feature (Proctor et al., 2003), and otherwise only occur following *noncorresponding* location trials (Hommel, 2004; Spapé et al., 2011; Wendt et al., 2006). A such, we assume that reversing the SR-mapping for an arrow target will not generalize to processing of the irrelevant stimulus location.

3.1.3 Domain-specific Conflict and Control

The predicted dissociation with a reversed SR-mapping provides us with a means to first confirm that Eriksen and Simon interference reflect different sources of conflict, rather than just the same source of conflict with different temporal dynamics (i.e. faster direct route activation for stimulus locations than for flanker arrows). Assuming that Eriksen and Simon interference is incurred via such different mechanisms, then we can also specify how different forms of cognitive control will be beneficial to preventing, attenuating, or resolving each type of response conflict. For example, some have proposed that response conflict can be prevented by a voluntary suppression of the direct route (de Jong, 1995; Duncan, 1978; Kornblum et al., 1990; Praamstra et al., 1999; Ridderinkhof, 2002; Shaffer, 1965; Stoffels, 1996b; Stürmer et al., 2002, 2007; Vu & Proctor, 2004). Assuming that Simon and Eriksen interference reflect different sources of conflict in a dual route model, suppressing the direct route would be beneficial to preventing Simon interference, but not to preventing Eriksen interference.

Alternatively, a more general reduction to any response activation (c.f. Band & van Boxtel, 1999; Band et al., 2003; Jennings & van der Molen, 2005) might reduce both Simon and Eriksen interference, and would be particularly beneficial in incompatible tasks to allow sufficient time to identify the correct response. Such a mechanism fits with accounts of the speed-accuracy trade-off (see Bogacz et al., 2010, for a review), and the idea that we are able to control the level of response activation even before a stimulus has been presented (Niemi & Näätänen, 1981; see also Brown & Heathcote, 2005; Jahfari et al., 2012; Jennings & van der Molen, 2005; Hanes & Schall, 1996). Event Related Potentials (ERPs) have already provided measures associated with automatic response activation and its inhibition, which can be used to assess the domain-specificity of conflict resolution with different sources of response conflict.

3.1.4 Measuring Conflict and Control with ERPs

Automatic response activation associated with response conflict has often been assessed using the lateralized readiness potential (LRP; Coles 1989; de Jong et al, 1988; Gratton et al., 1988, 1992). The LRP is a difference wave that compares the relative amount of contra- and ipsilateral motor cortex activation in left- versus right-hand responses, representing the build-up of preferential response activation of correct vs. incorrect response channels. As well as illustrating the build-up of contralateral activation that peaks just before response execution, the LRP has identified early preferential response activation toward the incorrect response in incongruent Eriksen trials (e.g. Gratton et al., 1988, 1992; Heil et al., 2000, Kopp et al., 1996; van 't Ent, 2002; Wascher et al., 1999; Willemssen et al., 2004).

More recent studies have demonstrated the advantages of assessing individual contributions of motor cortex activity contralateral and ipsilateral to the response (Praamstra & Seiss, 2005; Vidal et al., 2003; Yordanova et al., 2004). Furthermore, analyses using Laplacian transformations of recordings above the motor cortices in choice reaction time (RT) tasks suggest that prior to the response contralateral negativity reflects activation of the correct response and ipsilateral positivity reflects inhibition of the incorrect response (Burle et al., 2004; Meckler et al., 2010; Praamstra & Seiss, 2005; van der Laar et al., 2012, 2014; Vidal et al., 2003). Based on this knowledge, we can use ERPs recorded over the motor cortices to compare relative polarity contralateral and ipsilateral to the response with each type of interference. Theoretically, response conflict should be reflected by reduced negativity contralateral to the response and increased negativity ipsilateral to the response, relative to non-conflict conditions. This prediction allows us to assess the temporal dynamics of conflict and its resolution in contralateral and ipsilateral motor cortex activity.

Using waveforms recorded over motor cortices to assess the temporal dynamics of response conflict with Simon interference is less straightforward, as previous studies with lateralized stimuli have demonstrated contamination of activity recorded over motor areas by early stimulus-locked asymmetries at parietal/occipital electrodes (Praamstra & Oostenveld, 2003; Valle-Inclan, 1996; van der Lubbe et al., 2001; Wascher & Wauschkuhn, 1996). However, these parietal asymmetries around 200 ms following stimulus onset (an early contralateral N2) are assumed to reflect spatial attention-related processes (Eimer,

1996; Luck & Hillyard, 1994; Praamstra and Oostenveld, 2003). Similarly, contralateral N2 at central areas has been interpreted to reflect spatial attentional-motor-related processing (Leuthold & Schröter, 2006; Praamstra and Oostenveld, 2003). Our previous study (Mansfield et al., 2013) found that posterior asymmetries in the EEG had subsided prior to the interval associated with Eriksen interference, which is what we aim to reassess in the current study with mixed interference for both a compatible and an incompatible SR-mapping.

If Eriksen and Simon interference are resolved in a domain-specific manner, then each type of interference should be dissociable in ERP measures of cognitive control. Online cognitive control is consistently associated with midline N2, which peaks 200-350 ms following presentation of the task-relevant stimulus (see Folstein & van Petten, 2008, for a review). Notably, effects at midline N2 can have either a frontal or a central-parietal maximum, and as such Folstein and van Petten (2008) concluded that the more central or posterior N2 likely reflects mismatch, whereas the more anterior N2 likely reflects mechanisms of control triggered by conflict. Many studies have interpreted an enhanced midline fronto-central N2 to reflect inhibition of an active incorrect response (Carriero et al., 2007; Falkenstein et al., 1999, 2002; Heil et al., 2000; Kopp et al., 1996; van Boxtel et al., 2001), and others have suggested that it reflects detection of response conflict (Carter & van Veen, 2007; Donkers & van Boxtel, 2004; Nieuwenhuis et al., 2003; van Veen & Carter, 2002; Yeung et al., 2004). According to the latter interpretation, an enhanced N2 with either Eriksen or Simon interference could be interpreted to suggest that response conflict is of greater magnitude with one type of interference. But comparison with performance and measures of response conflict over motor cortices could help to assess whether such an enhancement reflects conflict or its resolution.

Previously, we found that midline N2 was insensitive to both the type of interference and the amount of response conflict (Mansfield et al., 2013) when Simon and Eriksen interference were presented in separate experimental blocks, but a later midline/contralateral component (N350) was associated with resolution of Eriksen interference. However, in the current study we present both types of interference within blocks, implying that participants will not be able to predict and prepare to resolve specific types of interference. As such, we expect a greater amount of response conflict and online

control in the current experiment. As suggested, resolving Simon interference might involve suppression of the direct route (c.f. Kornblum et al., 1990), and be reflected in an enhanced anterior midline N2 with incongruent locations; whereas resolving Eriksen interference might be associated with a general response suppression (c.f. Band & van Boxtel, 1999), to allow sufficient time to select the appropriate response, and be reflected in a late enhanced N2 with incongruent flankers.

When expecting Simon interference, it might be beneficial to suppress direct-route priming even prior to stimulus presentation, which should attenuate Simon effects more than Eriksen effects and potentially account for the absence of Simon interference reflected in lateralized N2 in Mansfield et al. (2013). But with mixed interference, participants cannot predict location interference before stimulus presentation. This implies that resolving Simon interference might depend upon detection of response conflict, which should be reflected in an enhanced N2 with location conflict. Alternatively, participants might be able to suppress SR-priming even before response conflict has been detected, based on fast detection of specific stimulus features, such as unilateral stimuli. If so, we can expect midline N2 to be enhanced with all Simon stimuli (congruent and incongruent) compared to Eriksen stimuli. A candidate ERP component that might reflect fast detection of relevant stimulus attributes is the visual N1 component, which has been interpreted to reflect visual discrimination processes (Vogel & Luck, 2000). Eriksen interference, on the other hand, we expect to reflect mostly indirect-route processing, and as such should not be attenuated by suppression of the direct route. However, Eriksen interference could potentially be resolved by increased control via the indirect route, such as focusing more on the target and ignoring the flankers, or a general suppression of response activation (c.f. Band & van Boxtel). Such a general suppression would be particularly beneficial in incompatible tasks when identifying the correct response is more difficult, and would predict an enhanced N2 in incompatible compared to compatible blocks, as well as generally slower responses.

3.1.5 Hypotheses

We hypothesize that conflict resolution by cognitive control is flexible and domain specific (c.f. Egner, 2008), and that interference is both incurred and resolved differently for Eriksen compared to Simon interference (c.f. Mansfield et al, 2013). We use two lines of

investigation to assess this hypothesis: first we aim to confirm that Eriksen and Simon interference represent different sources of response conflict; and then we aim to assess the control mechanisms that resolve each type of interference. We use performance measures and contralateral/ipsilateral activity over motor cortices to assess the magnitude and temporal dynamics of response conflict, predicting response conflict to be associated with reduced contralateral negativity and enhanced ipsilateral negativity; and we use performance and midline ERPs to assess cognitive control.

If Eriksen and Simon interference reflect different sources of conflict incurred via different routes of a dual-route model (c.f. Mansfield et al., 2013), then in compatible blocks we should replicate our earlier finding of smaller effects for Simon compared to Eriksen interference, and in incompatible blocks Eriksen effects should remain intact but Simon effects should appear to reverse. This is because in incompatible blocks, incongruent flanker arrows should be subject to the same reversed SR-mapping (e.g. left=right) via the indirect route, and so facilitate the incorrect response. In contrast, stimuli presented to the left of fixation will continue to activate a left-hand response via the direct route, such that in relation to the target arrow interference by locations will be reversed in incompatible blocks. Furthermore, activity recorded over contralateral/ipsilateral motor cortices should demonstrate similar congruency patterns of response activation for both SR-Mapping tasks for Eriksen interference, but reversed congruency patterns for Simon interference.

Importantly, here we present both types of interference in the same experimental blocks, which allows us to compare cognitive control strategies with each type of interference. Specifically, we expect early detection of potential location interference (reflected in enhanced midline N1), followed by suppression of the direct route (reflected in an enhanced N2) with all Simon stimuli. If the control at N2 is sensitive to the amount of conflict in each trial type, then N2 should show a similar pattern of effects as performance, with enhanced amplitudes for the trials demonstrating the most conflict (in performance and motor cortex activity). If Eriksen interference is resolved by a general suppression to response activation, then responses should be generally slower in Eriksen trials and a late midline N2 should be enhanced with incongruent flankers. Comparing two types of interference with different SR-mapping instructions also allows us to assess the flexibility of cognitive control depending upon task goals. If a general suppression of response activation

is applied to reduce response conflict with the reversed SR-mapping, then in incompatible blocks responses should be generally slower (without effects on accuracy), and all interference effects should be reduced, reflected mainly in detriments to non-conflict trials.

3.2 METHOD

3.2.1 Participants

Seventeen psychology students gave their informed consent to participate in this study as part of a course requirement. Three participants were excluded from analyses; one due to failure to complete the experiment, and two participants due to excess artifact in the electrophysiological data. The remaining fourteen participants (12 women and 2 men) were right-handed, had no history of neurological disorder, and were not taking medication or drugs known to influence performance. Their ages ranged from 18 to 29 (mean 20 years). All participants had normal or corrected-to-normal vision and gave informed consent before taking part.

3.2.2 Apparatus

The participants were seated in a comfortable chair in a dimly lit, ventilated, soundproofed, and electrically-shielded room. A 14-inch monitor was placed at a distance of 130 cm in front of the participants' eyes. Responses were measured and A/D-converted on-line from Kyowa ML-20KA zero-displacement force transducers, which were built into two custom-made hand-supports at the point where the participant's index finger rested. These measured both the reaction times (the moment at which a response reached two percent of the participant's maximum force) and the pressure for each trial.

3.2.3 Tasks

The experiment consisted of two tasks measured between experimental blocks: compatible or incompatible responses to the direction of a central arrow (see figure 3.1). In *compatible-mapping* blocks participants had to respond to the direction of the arrow by pressing the index finger of the corresponding hand. In *incompatible-mapping* blocks participants had to respond to the direction of the arrow using the index finger of the opposite hand.

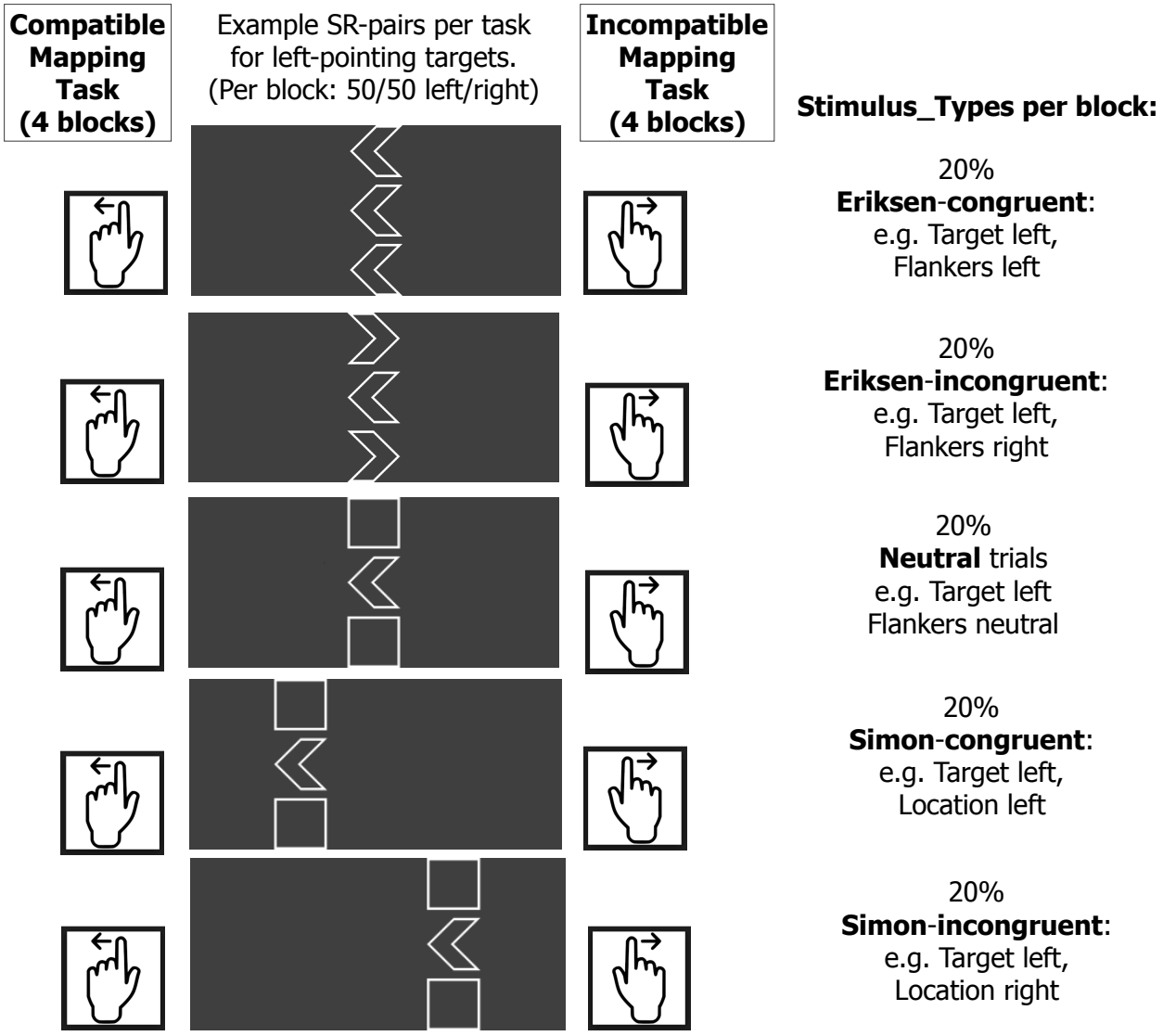

Figure 3.1. Schematic overview of the five stimulus-types and associated response in each SR-mapping task (compatible/incompatible), here for left-pointing targets only (50% of trials per block). Note that in incompatible-mapping tasks, interference (Simon or Eriksen) that is incongruent with the target arrow is congruent with the correct response. "Congruency" refers to the relationship between interference and target (not response).

3.2.4 Stimuli and Design

Each trial consisted of an array of three vertically presented white stimuli against a dark gray background, of which the central arrow was always the target (see figure 3.1). The flanker-stimuli above and below the target were either congruent or incongruent arrows for Eriksen interference, or squares for neutral trials and Simon interference. The neutral trials (centrally-presented with square flankers) functioned as a control condition for both Simon and Eriksen interference. Each stimulus array measured 2.5 x 9 cm and had a visual angle of

1.1°. In the Simon conditions the stimuli were presented 3 cm to the left/right of fixation (1.3°). There were 10 stimuli, split into 5 conditions (Eriksen congruent 20%, Eriksen incongruent 20%, Simon congruent 20%, Simon incongruent 20%, and neutral 20%), and 2 target directions (left 50%, right 50%). All stimulus-types were presented together in mixed lists of 200 trials. Each stimulus-list (block) was made up of 4 sub-lists of 50 randomized stimuli, such that each stimulus-type occurred just as often in the beginning, end, or middle of the complete list.

3.2.5 Procedure

Prior to the experiment, maximum force was measured (at least three times for each hand, in order to calculate an average maximum force per subject). Half of the participants performed the compatible-mapping task first, and the other half performed the incompatible-mapping task first. The experiment consisted of eight experimental blocks (four per SR-Mapping), and each task was preceded by two training blocks of 30 trials. During the training session, participants received feedback concerning their performance ("too weak", "wrong hand", "too late", or "good"). A trial began with the presentation of a white fixation cross in the center of the screen for 2000 – 4000 ms (random variable ITI), which was replaced by the stimulus array for 150 ms. The fixation cross was always present, apart from during the 150 ms target-presentation, and the response-deadline was set at 1200 ms. Participants were asked to ignore the flankers and the location of the stimuli, to keep their eyes on fixation, and to respond as quickly and accurately as possible, by pressing the appropriate index finger briefly but firmly. In order to reduce artifact in the electrophysiological data, participants were asked to sit as still as possible. Each block consisted of 200 trials, lasted approximately 10 minutes, and was followed by a short break. The entire experimental session took approximately 3,5 hours.

3.2.6 Electrophysiological Recording

EEG was recorded from Fz, FCz, Cz, Pz, C3, and C4, using Beckman Ag/AgCl electrodes with a diameter of 8 mm, affixed to the scalp with Grass EC-2 electrode paste (amplified 20,000 times). Horizontal EOG was recorded from the outer canthi of both eyes, and vertical EOG from above and below the right eye, using Beckman Ag/AgCl electrodes with a diameter of 2 mm (affixed with Signa gel), and amplified 10,000 times. Bipolar EMG was measured at the

forearm flexor of both arms, with a reference placed on the back of the right-hand, using Beckman Ag/AgCl electrodes with a diameter of 2 mm, affixed with Signa gel. EMG signals were amplified on-line 1000 times, high-pass filtered at 20Hz, then full-wave rectified, then low-pass filtered at 50Hz; this procedure yielded EMG envelope activity.

3.2.7 Electrophysiological Data-Analysis

Data analysis was performed on ERPs using the Brain Vision Analyzer software package. Signals were referenced to average mastoids and then band filtered from 0.1 Hz to 15 Hz (48 dB/octave roll-off). Horizontal EOG was calculated (EOGr-EOGl) and filtered with a high cut-off of 5Hz in order to facilitate detection of horizontal eye-movements. EEG was segmented (from 200 ms before until 1000 ms after target onset), and all EEG channels were corrected for blinks, using the regression technique by Gratton and Coles (1983). In order to minimize the influence of eye movements on ERPs, following visual inspection, all epochs containing horizontal EOG with a difference of more than 20 microvolts within any 150 ms window were rejected. Subsequently, segments with artifacts on the remaining electrodes were rejected automatically with a 100 microvolt difference criterion (within the entire segment). Baseline correction was performed according to the 100 ms prior to target onset, and epochs were averaged for each subject per condition. A minimum of 21 trials per participant per condition was required for a participant to be included in the grand average. For visual inspection purposes, LRPs were calculated according to the formula:
[C4(left-response)-C3(left-response)+C3(right-response)-C4(right-response)] / 2.
Correct response activation was therefore represented by negative amplitudes. The same calculation was also made for left and right EOG. In order to rule out the possibility that minute eye movements toward the stimulus location contaminated analyses of motor cortex activity, correlations were calculated between LRPs and LEOGs between 250-300 ms for all Simon conditions, which confirmed that LRPs were not influenced by horizontal EOG (all *p*s>.1). For analyses of motor cortex activity, waveforms were calculated separately for C3/C4 contralateral and ipsilateral to the response hand (c.f. Yordanova et al., 2004).

3.2.8 Statistical Analysis

A valid response required the pressure on the force transducer to achieve at least 15% of the participant's maximum force; for valid trials, RT was calculated as the moment at which the

pressure reached 2% of the participant's maximum force. Only correct responses between 200 ms and 1000 ms were included in analyses of RTs. We used RTs, error proportions and contralateral/ipsilateral motor cortex activity to measure response conflict, and midline N1 and N2 to assess the control mechanisms associated with resolving response conflict. All analyses of behavioral and electrophysiological measures were repeated measures ANOVAs including the within-subjects factors SR-Mapping (compatible/incompatible), Interference_Type (Simon/Eriksen) and Congruency (congruent/incongruent). Analyses of lateralized ERPs initially included the factor Lateralization (contralateral/ipsilateral), in order to assess whether interference effects differed according to hemisphere.

Analysis of performance in compatible blocks aimed to replicate the finding of larger interference effects for Eriksen compared to Simon interference, but now in mixed interference blocks. To this goal, we assessed error proportions and RTs with the factors, Interference_Type (Eriksen / Simon) and Congruency (Congruent / Incongruent). Subsequently, as an additional test of the domain-specificity of conflict in Eriksen/Simon tasks, we assessed the effects of reversing the SR-mapping instruction on each type of interference. To this end, all measures were assessed including the factor SR-Mapping (compatible / incompatible), and then broken down to identify the direction and magnitude of Eriksen/Simon interference with a reversed mapping.

Neutral trials served as a control condition: to ensure that interference by incongruent flankers/locations did not reflect visual conflict or mismatch; and for disentangling effects of SR-Mapping from the effects associated with unpredictable Simon/Eriksen interference. As such, paired t-tests were performed comparing congruent/incongruent interference to neutral trials in compatible blocks (to rule out visual conflict effects), and comparing neutral trials between compatible and incompatible mapping tasks (to assess the SR-Mapping effect).

3.4 RESULTS

Means for all measures are reported in Table 3.1. Congruency is reported according to stimulus congruency (between target and interference), which implies that with the incompatible mapping, stimulus-incongruent interference is congruent with the response (see figure 3.2).

	LRP:105	ContraN100	IpsiN100	MidN1	ContraN250	IpsiN250	ContraN300	IpsiN300	MidN2	RT	Errors
Cmap_ECong	-.09	.17	.27	.83	3.10	3.32	3.22	4.83	.68	404	.017
Cmap_Einco	-.19	.67	.87	1.20	4.48	4.18	3.92	4.16	1.03	453	.073
Cmap_SCong	1.13	.26	-.91	-1.68	3.37	3.99	1.55	3.90	-1.01	394	.016
Cmap_SInco	-1.24	-.74	.48	-1.61	3.55	3.37	2.35	3.21	-.89	426	.072
Cmap_Neutral	.11	.23	.13	-.81	3.91	3.85	2.57	4.02	-.58	400	.029
Imap_Neutral	-.05	.16	.14	.07	4.03	4.39	2.63	3.78	.14	422	.037
Imap_ECong	.21	.50	.30	.80	3.23	3.45	3.00	3.95	1.12	426	.044
Imap_EInco	.37	.89	.61	.88	3.50	3.48	3.28	4.15	1.01	450	0.40
Imap_Scong	-1.10	-.35	.72	-1.65	3.56	3.48	2.29	2.37	-1.10	444	.078
Imap_SInco	1.12	.56	-.51	-1.41	3.06	3.43	1.31	3.00	-.88	429	.033

Table 3.1. Means per Stimulus_Type with each SR-Mapping for all measures.

3.4.1 Behavioral Results

Behavioral data are depicted in figure 3.2 with SR-pairs for right-hand responses, to illustrate the coding for the factor Congruency (between target and interference), with lines depicting the direction and magnitude of Eriksen/Simon effects in each SR-mapping task.

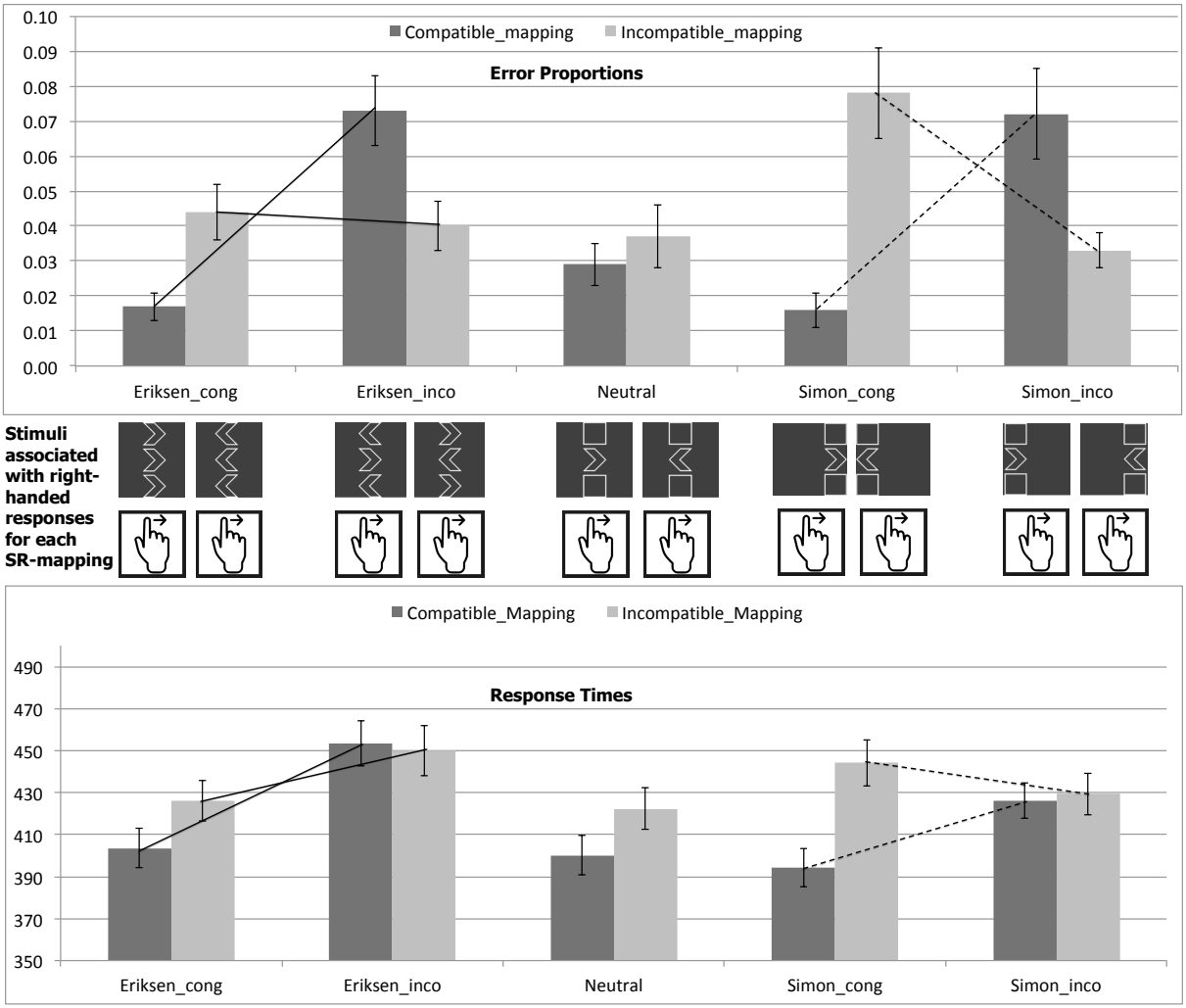

Figure 3.2. Mean errors (top) and RTs (bottom) comparing compatible/incompatible mapping instructions according to stimulus-stimulus congruency between target arrow and flankers/location, accompanied by example stimulus-response pairings for right-hand responses (middle). The direction and magnitude of interference effects within each SR-mapping task is highlighted by solid lines for Eriksen interference and dashed lines for Simon interference. Note that with the incompatible mapping, interference that is incongruent with the target could facilitate and/or inhibit the correct response, depending on the degree to which such interference is subject to both direct route response priming and to generalization of the SR-mapping instruction (e.g. left=right) via the indirect route.

3.4.1.1 Comparisons with Neutral Trials

Neutral trials were used for control analyses. Firstly, neutral trials were compared to interference trials in compatible blocks to confirm the extent to which interference reflected visual processing. For Eriksen interference, neutral flankers were similar to congruent flankers in RTs (t = 1.51, p = .155) but marginally less accurate (t = 2.16, p = .05), but incongruent flankers resulted in longer RTs (t = 12.36, p < .001) and more errors (t = 6.70, p < .001) than neutral flankers. For Simon interference, congruent locations resulted in marginally shorter RTs (t = 1.99, p = .069) and marginally less errors (t = 2.16, p = .05) than

neutral trials, and incongruent locations resulted in significant detriments to both RTs (t = 8.02, p < .001) and accuracy (t = 5.16, p < .001). These comparisons with neutral trials in compatible blocks confirmed that interference by both incongruent flankers and incongruent locations did not reflect purely visual differences. Secondly, neutral trials were used to assess the magnitude of the SR-Mapping effect. Paired t-tests confirmed that neutral trials were slower in incompatible blocks (t(13) = 3.57, p = .003), but there was no SR-Mapping effect in error proportions (t< 2.0, p > .2), suggesting a shift toward accuracy over speed in incompatible blocks.

3.4.1.2 Compatible SR-Mapping

Replicating previous findings (Mansfield et al. 2013), in compatible blocks Eriksen interference was larger than Simon interference on analysis of RTs (Interference_Type x Congruency, F(1,13) = 12.1, p=.004, η_p^2 = .482), but on analysis of errors the Congruency effect was equal for Simon and Eriksen interference (interaction, F<1; Congruency, F(1,13) = 38.1, p<.001, η_p^2 = .746). Simple effects confirmed that the Congruency effect was also present on separate analysis of Eriksen interference on RTs (Congruency, F(1,13) = 97.5, p<.001, η_p^2 = .882) and errors (F(1,13) = 42.4, p<.001, η_p^2 = .765), and on separate analysis of Simon interference on RTs (F(1,13) = 82.5, p<.001, η_p^2 = .864) and errors F(1,13) = 23.0, p<.001, η_p^2 = .639).

3.4.1.3 Reversing the SR-Mapping

Confirming the SR-Mapping effect, responses were generally slower in incompatible blocks (437 ms) compared to compatible blocks (419 ms), F(1,13) = 7.6, p=.016, η_p^2 = .369, but there was no difference in errors (compatible = 4.4%, incompatible = 4.9%; F<1). As expected, Eriksen and Simon interference demonstrated a different pattern of effects with an incompatible SR-mapping, supported by a 3-way interaction in errors (F(1,13) = 8.8, p=.011, η_p^2 = .403) and RTs (F(1,13) = 5.7, p=.033, η_p^2 = .304), which we then broke down per Interference_Type.

Eriksen interference. Reversing the SR-mapping eliminated Eriksen effects in errors and reduced effects in RTs (SR-Mapping x Congruency: errors, F(1,13) = 43.7, p<.001, η_p^2 = .771; RTs, F(1,13) = 22.8, p<.001, η_p^2 = .637). Simple effects in incompatible blocks revealed that incongruent flankers were still slower than congruent flankers in RTs (F (1,13) = 43.0,

p<.001, η_p^2 = .768), but the Eriksen interference effect was eliminated in errors (ns, see figure 3.2, top left).

Simon interference. Reversing the SR-mapping reversed Simon congruency effects in errors and RTs (SR-Mapping x Congruency: errors, $F(1,13) = 27.4$, p<.001, η_p^2 = .678; RTs, $F(1,13) = 44.8$, p<.001, η_p^2 = .775). Simple effects confirmed that with an incompatible mapping locations that were congruent with the target arrow resulted in the most errors ($F(1,13) = 21.1$, p=.001, η_p^2 = .619) and the slowest RTs ($F(1,13) = 8.2$, p=.013, η_p^2 = .388).

In sum, Eriksen interference effects were significantly larger than Simon interference effects on analysis of RTs, but not errors, replicating previous findings with blocked interference types (Mansfield et al., 2013). Reversing the SR-mapping had differential effects on Eriksen and Simon interference, such that in incompatible blocks, interference effects were reduced for incongruent flankers but reversed for incongruent locations. In other words, irrelevant locations continued to facilitate the response corresponding to the stimulus location, and did not demonstrate automatic generalization of the task instruction (e.g. left=right) to the irrelevant spatial dimension.

3.4.2 Lateralized Readiness Potentials

For visual inspection purposes, LRPs are illustrated in Figure 3.3 (top panel), for compatible (left) and incompatible blocks (right), and mean amplitudes are reported in Table 3.1. As can be seen in Figure 3.3, Simon stimuli were associated with a very early bipolar deflection, peaking at around 105 ms following stimulus onset. Interestingly, this deflection suggested early activation of the *opposite* response with a compatible mapping (i.e. positive amplitudes for congruent stimuli and negative amplitudes for incongruent stimuli). With the incompatible mapping, the same early deflection was reversed, such that congruent locations demonstrated an early negative deflection, and incongruent locations demonstrated an early positive deflection. Later on in LRPs for incompatible mapping blocks (around 300 ms), there was also a visible delay to correct response activation with congruent locations, reflecting the performance detriments found for these trials. Eriksen interference was visible in a later deflection (around 250 ms) in compatible blocks, but this deflection was absent in incompatible blocks. Subsequently, response conflict was assessed for contralateral and ipsilateral waveforms.

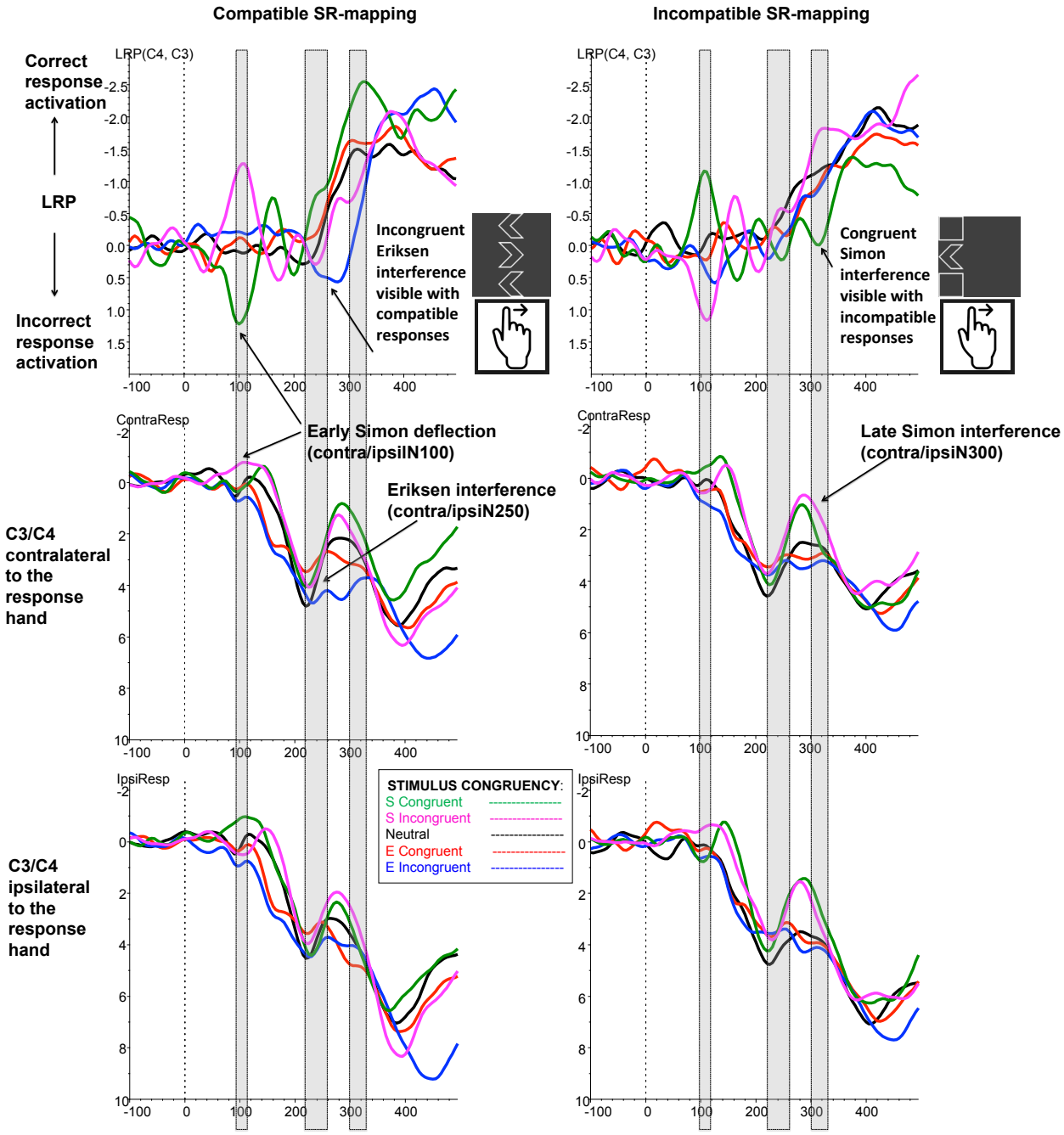

Figure 3.3. Stimulus-locked (time=0) ERPs above motor areas (C3/C4) with the compatible mapping (left) and incompatible mapping (right), according to interference-type (Simon=S, Eriksen=E) and congruency between interference and target (see legend). Top panel: Lateralized Readiness Potentials (LRPs), depicting the build-up of activation in favor of the correct response. Middle panel: activation contralateral to the correct response hand. Bottom panel: activation ipsilateral to the correct response hand. Shaded areas depict measurement windows for contralateral/ipsilateral N100, N250 and N300.

3.4.3 Contralateral and Ipsilateral Motor Cortex Activation

Figure 3.3 depicts stimulus-locked activity recorded over C3/C4 for individual contributions of contralateral (middle panel) and ipsilateral (lower panel) motor cortex activation with a compatible (left) and incompatible (right) SR-mapping, and mean amplitudes per condition are reported in Table 2. Following visual inspection of grand averaged waveforms, components were assessed in three separate intervals; the first corresponding to the early deflection found in LRPs with Simon interference (contra/ipsiN100: 95-115 ms), the second corresponding to visible Eriksen interference (contra/ipsiN250: 220-260 ms), and the third corresponding to later interference with Simon stimuli (contra/ipsiN300:300-330 ms). Paired t-tests for neutral trials revealed that all 3 components were insensitive to the SR-Mapping effect alone, for both contralateral and ipsilateral activity (all *t*s<2, *p*s>.05).

3.4.3.1 Early Asymmetry with Simon Interference (Contralteral/Ipsilateral N100)

In order to investigate the early deflection seen in LRPs for Simon interference, we analyzed contra- and ipsilateral activity in the interval around 105 ms following stimulus onset. Careful inspection of Figure 3.3 suggested that the early LRP deflection with Simon stimuli coincided with visible increased negativity *ipsilateral* to the stimulus location. In compatible blocks this negative deflection was *ipsilateral* to the response with congruent locations and *contralateral* to the response with incongruent locations. In incompatible blocks the same negative deflection was enhanced *contralateral* to the response for congruent locations, and *ipsilateral* to the response for incongruent locations. The omnibus ANOVA revealed a four-way interaction between Lateralization, SR-Mapping, Interference_Type and Congruency, ($F(1,13) = 16.0$, $p=.002$, $\eta_p^2 = .552$). Subsequently separate analyses were performed for contra- and ipsilateral activation.

Contralateral to the response, the pattern resulted in a three-way interaction ($F(1,13) = 8.1$, $p=.014$, $\eta_p^2 = .383$). As expected, separate analysis of Eriksen interference revealed no effects (all *p*s>.05). Separate analysis of Simon interference confirmed an interaction (SR-Mapping x Congruency, $F(1,13) = 16.9$, $p=.001$, $\eta_p^2 = .565$), supported by simple effects, such that with a compatible mapping contralateral N100 was enhanced for incongruent locations ($F(1,13) = 9.8$, $p=.008$, $\eta_p^2 = .430$), but with the incompatible mapping for congruent locations ($F(1,13) = 11.3$, $p=.005$, $\eta_p^2 = .464$).

Ipsilateral to the response, the pattern also resulted in a three-way interaction, (F(1,13) = 9.6, p=.009, η_p^2 = .424). Separate analysis of Eriksen interference revealed a *positive* deflection that was largest for incongruent flankers (Congruency, F(1,13) = 11.2, p=.005, η_p^2 = .464), but neither the SR-Mapping effect nor the interaction effect approached significance (ps>.1). Separate analysis of Simon interference confirmed the interaction (SR-Mapping x Congruency, F(1,13) = 15.5, p=.002, η_p^2 = .544), supported by simple effects, such that with a compatible mapping ipsilateral N100 was maximal for congruent locations (F(1,13) = 18.7, p=.001, η_p^2 = .590) but with an incompatible mapping for incongruent locations (F(1,13) = 10.0 p=.007, η_p^2 = .435).

3.4.3.2 Contralateral/Ipsilateral N250

Inspection of figure 3.3 revealed differences between congruent and incongruent Eriksen trials (220-260 ms) contralateral to the response with the compatible SR-mapping, which were diminished with an incompatible SR-Mapping. This pattern resulted in a three-way interaction in the omnibus ANOVA between Lateralization, SR-Mapping and Congruency (F(1,13) = 8.4, p=.012, η_p^2 = .393). Subsequently, separate analyses were conducted per hemisphere, to assess individual contributions of contralateral and ipsilateral motor cortex activity.

Separate analyses per hemisphere confirmed the absence of any effects ipsilateral to the response (all Fs< 3.4, ps>.09). However, contralateral to the response, Congruency effects were larger with a compatible mapping than with an incompatible mapping (SR-Mapping x Congruency, F(1,13) = 6.4, p=.025, η_p^2 = .329), and larger for Eriksen interference compared to Simon interference (Interference_Type x Congruency, F(1,13) = 5.1, p=.042, η_p^2 = .282). This interaction was then broken down further to confirm the absence of any interference effects for Simon interference (all ps>.1), while for Eriksen interference, incongruent flankers resulted in a reduced contralateral N250 compared to congruent flankers (F(1,13) = 13.3, p=.003, η_p^2 = .505), and a marginal interaction between SR-Mapping and Congruency (F(1,13) = 4.4, p=.057, η_p^2 = .251). Paired t-tests confirmed that the reduction to contralateral N250 with Eriksen interference was significant with the compatible mapping (t=4.0, p=.002) but not with the incompatible mapping (t=1.5, p=.161).

3.4.3.3 Contralateral/Ipsilateral N300

This late component was maximal contralateral to the response ($F(1,13) = 38.0$, $p<.001$, $\eta_p^2 = .745$), and larger with Simon interference compared to Eriksen interference ($F(1,13) = 16.4$, $p=.001$, $\eta_p^2 = .558$). Furthermore two significant 3-way interactions between Lateralization x SR-Mapping x Interference_Type ($F(1,13) = 6.4$, $p=.026$, $\eta_p^2 = .328$) and Lateralization x SR-Mapping x Congruency ($F(1,13) = 27.2$, $p<.001$, $\eta_p^2 = .676$), and a trending 4-way interaction ($F(1,13) = 4.3$, $p=.058$, $\eta_p^2 = .249$) indicated the relevance of separate analyses per hemisphere.

Contralateral to the response, N300 was enhanced for Simon interference ($F(1,13) = 19.0$, $p=.001$, $\eta_p^2 = .594$). Furthermore, the direction of the interference effect depended upon the SR-Mapping (SR-Mapping x Congruency, $F(1,13) = 13.5$, $p=.003$, $\eta_p^2 = .510$), and the critical 3-way interaction was marginal (SR-Mapping x Interference_Type x Congruency, $F(1,13) = 4.6$, $p=.051$, $\eta_p^2 = .261$). Subsequently, we conducted separate analyses per SR-Mapping. With a compatible mapping, contralateral N300 was largest with Simon interference ($F(1,13) = 16.2$, $p=.001$, $\eta_p^2 = .554$), and reduced with *incongruent* interference ($F(1,13) = 5.0$, $p=.044$, $\eta_p^2 = .278$), but insensitive to the interaction between Interference_Type and Congruency ($F<1$). However, with a reversed SR-mapping, an interaction (Interference_Type x Congruency, $F(1,13) = 6.9$, $p=.021$, $\eta_p^2 = .348$) suggested that a reduction to contralateral N300 with *congruent* interference was specific to Simon interference. Paired t-tests confirmed that with an *incompatible* SR-Mapping, contralateral N300 was reduced with *congruent* compared to incongruent locations ($t=3.2$, $p=.008$), but not with congruent compared to incongruent flankers ($t=1.1$, $p=.305$).

Ipsilateral to the response, N300 was also largest with Simon interference ($F(1,13) = 11.9$, $p=.004$, $\eta_p^2 = .477$), and interference effects depended upon SR-Mapping (SR-Mapping x Congruency, $F(1,13) = 5.0$, $p=.044$, $\eta_p^2 = .276$). In compatible blocks, ipsilateral N300 was greater with Simon compared to Eriksen interference ($F(1,13) = 5.4$, $p=.037$, $\eta_p^2 = .293$), but neither the effect of Congruency ($F(1,13) = 3.7$, $p=.078$, $\eta_p^2 = .220$) nor the interaction ($F<1$) were reliable. Similarly, in incompatible blocks, ipsilateral N300 was also larger with Simon compared to Eriksen interference ($F(1,13) = 13.0$, $p=.003$, $\eta_p^2 = .501$), but neither the effect of Congruency ($F(1,13) = 2.2$, $p=.160$, $\eta_p^2 = .146$) nor the interaction ($F<1$) were reliable.

In sum, activity recorded over motor cortices contralateral and ipsilateral to the response was assessed in three intervals. A very early asymmetry with Simon interference was always maximal *ipsilateral* to the *stimulus* location. Contralateral N250 was reduced only with Eriksen interference in compatible blocks, but this effect was eliminated in incompatible blocks. Contralateral N300 was maximal with Simon interference, and in compatible blocks this late component was reduced with all *incongruent* interference, but in incompatible blocks reduced only with *congruent* locations.

3.4.4 Midline N1

Grand averaged midline ERPs at FCz are illustrated in figure 3.4. Following inspection of grand averaged waveforms, N1 was scored as the mean amplitude from 130-160 ms following stimulus onset. As predicted, midline N1 was larger with Simon interference compared to Eriksen interference ($F(1,13) = 42.7$, $p<.001$, $\eta_p^2 = .766$), but was not affected by Congruency, SR-Mapping, or any interactions (all Fs<1). In order to rule out stimulus-conflict effects, we compared both types of interference to neutral trials. We ran an additional ANOVA including the factors SR-Mapping and Stimulus_Type (all 5, depicted in figure 3.1), which confirmed the effect of Stimulus_Type ($F(2,23) = 21.4$, $p<.001$, $\eta_p^2 = .622$, Greenhouse-Geisser tests with corrected *df*s rounded off). Pairwise comparisons with neutral trials confirmed that N1 was consistently reduced with Eriksen interference (both *p*s<.001) and consistently enhanced with Simon interference (both *p*s<.05).

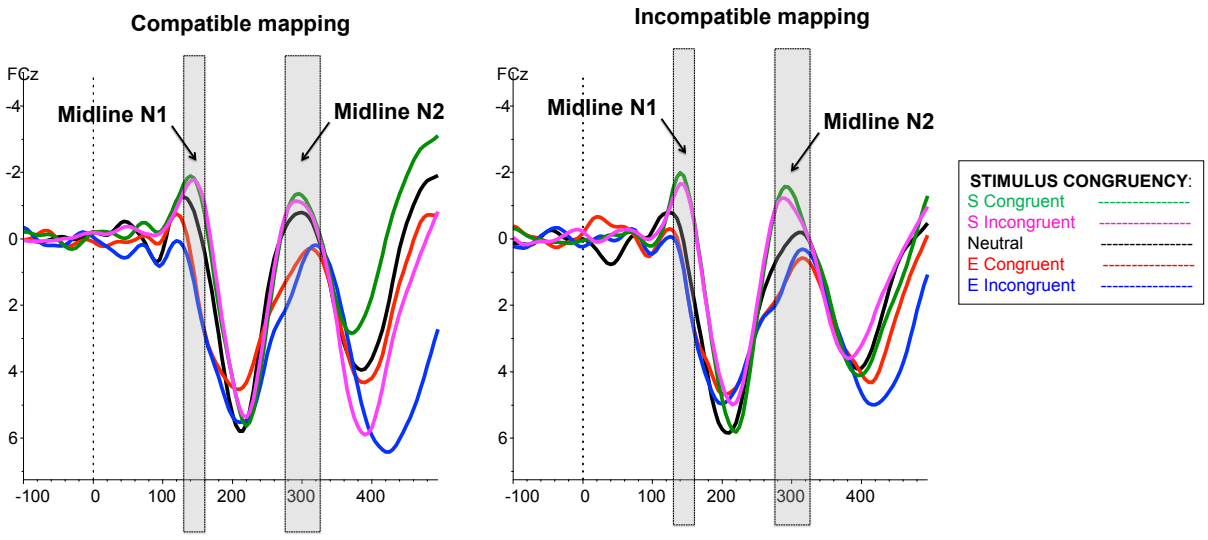

Figure 3.4. Midline waveforms recorded at FCz, for the compatible mapping (left) and incompatible mapping (right), according to interference-type and congruency between target and interference. Shaded areas depict measurement windows for midline N1 and midline N2.

3.4.5 Midline N2

Following inspection of grand averaged waveforms, N2 was scored as the mean amplitude from 275-325 ms following stimulus onset. As can be seen in figure 3.4, midline N2 was larger for Simon interference compared to Eriksen interference ($F(1,13) = 16.0$, $p=.002$, $\eta_p^2 = .552$). Despite the suggestion in figure 3.4 that N2 was slightly enhanced with congruent locations and incongruent flankers, neither Congruency, Mapping, nor any of the interactions were reliable (all *p*s>.1). In order to rule out stimulus-conflict effects, we compared both types of interference to neutral trials: we ran an additional ANOVA including the factors SR-Mapping and Stimulus_Type, which confirmed the effect of Stimulus_Type ($F(2,30) = 7.5$, $p=.001$, $\eta_p^2 = .366$, Greenhouse-Geisser tests with corrected *df*s rounded off). Pairwise comparisons with neutral trials confirmed that N2 was consistently reduced with Eriksen interference (both *p*s<.05), but the difference between neutral trials and Simon trials was unreliable (both *p*s>.1).

3.5 DISCUSSION

The primary goal of this study was to expand on limited evidence in favor of domain-specific cognitive control involved in the resolution of response conflict (c.f. Egner, 2008), for which we found support via two lines of analysis comparing Eriksen and Simon interference. Initially, we confirmed that Eriksen and Simon interference reflect different sources of conflict in a dual-route model. In support of this assumption, we replicated our earlier finding (which used a blocked-interference design), now using a mixed-interference design, of larger interference effects in RTs for Eriksen compared to Simon interference. Then we confirmed that reversing the SR-Mapping instruction had differential effects on Eriksen and Simon interference: Eriksen congruency effects were reduced; but Simon congruency effects appeared to reverse, such that locations that were incongruent with the target arrow facilitated the incompatible response. Comparisons of activity recorded over motor cortices for compatible and incompatible SR-mappings provided further support that automatic response activation by task irrelevant stimulus attributes takes effect via different routes for Eriksen vs. Simon interference, demonstrating the same pattern as the performance data. This dissociation suggests that Simon interference reflects only direct-route response activation, while Eriksen interference reflects primarily indirect-route response activation.

We hypothesized that in mixed interference blocks, detection of unilateral (Simon) stimuli is associated with a fast suppression of the direct route (automatic, unconditional response activation), preventing fast execution of the response corresponding to the stimulus-location. As expected, both midline N1 and N2 were enhanced with Simon interference compared to Eriksen interference, which we assume to reflect visual discrimination processes at N1 (Vogel & Luck, 2000) and increased cognitive control at N2 (c.f. Folstein & van Petten, 2008). Potentially, such increased online control with Simon stimuli accounts for smaller interference effects for Simon compared to Eriksen interference in compatible blocks. However, the reduction to Eriksen interference with the reversed SR-mapping was not accounted for by our ERP measures of online control, and requires careful comparison between available measures and with previous findings. To this goal, we will first assess the source of conflict for both types of interference, and then discuss the potential mechanisms of control involved in resolving unpredictable interference in the current study.

3.5.1 Confirming Two Routes to Response Conflict

In line with the Dimensional Overlap model and other dual-route models (e.g. de Jong, et al., 1994; Kornblum et al., 1990; Ridderinkhof et al., 1995), we hypothesized two routes to response conflict by irrelevant stimulus attributes (c.f. de Jong et al., 1994): automatic response activation via a fast, direct, unconditional route with Simon interference; and automatic response activation via a slower, indirect, conditional route with Eriksen interference. In the current task, both types of irrelevant interference (Eriksen and Simon) have dimensional overlap with both the target and the response (c.f. Kornblum et al., 1990; Egner, 2008), but to very different degrees. Specifically, irrelevant stimulus locations should have more dimensional overlap with the relevant response dimension (left/right response button) than with the relevant stimulus dimension (left/right pointing central arrow), but irrelevant flanker arrows should have more dimensional overlap with the relevant target arrow than with the response. As such, we expect Simon interference takes effect via the direct route and flanker interference takes effect via the indirect route. We tested this prediction by comparing interference effects with both a compatible and an incompatible SR-mapping on both performance and motor cortex activity.

We found effects of interference in ERP measures in three intervals: a very early asymmetry with unilateral stimuli, a contralateral N250, and a contralateral N300. As expected, we observed large early asymmetries with Simon interference, which have been shown to reflect contamination by activity at posterior electrodes (c.f. Praamstra & Oostenveld, 2003; Valle-Inclan, 1996; van der Lubbe et al., 2001; Wascher & Wauschkuhn, 1996). However, it is unlikely that early asymmetries contaminated measures of response conflict at C3/C4 in the N250 or N300 intervals because contra-/ipsilateral N250 was insensitive to Simon interference in both the current task and the study by Mansfield et al. (2013). Importantly, the interference conditions demonstrating the most response conflict in performance measures were associated with reduced negativity contralateral to the correct response, which is in line with the effects at contralateral N250 reported by Mansfield et al. (2013). As such, we interpret intervals of reduced contralateral negativity to reflect the temporal dynamics of response conflict with each type of interference.

3.5.2 Response Conflict with Simon Interference

Intriguingly, the very early deflection with unilateral stimuli was consistently maximal *ipsilateral* to the *stimulus* location, earlier and in the opposite direction to the expected effects. A similar but smaller component was also visible in LRPs at C3/4 and P3/4 depicted in Mansfield et al. (2013), in which Simon and Eriksen interference were presented in separate experimental blocks, but in the previous study this deflection was overpowered by subsequent activation *contralateral* to the stimulus location. Potentially, the inclusion of neutral flankers (squares) with Simon interference in the current study increased early asymmetries associated with visual processing, but this does not account for the unexpected direction of this deflection. One possible account is that the deflection in the current study might reflect the directing of spatial attention away from the stimulus, as participants were explicitly instructed not to move their eyes toward the stimulus location. Indeed, research into the electrophysiology of spatial attention has revealed very early components (P1 and N1) with a similar timing to be enhanced contralateral to the focus of attention (Griffin et al, 2002, Luck et al., 1990). In other words, this very early component possibly reflects inhibition of overt eye movements toward the stimulus, and we are confident that such early asymmetry did not contaminate the later measures of motor activity that we interpret to reflect response conflict.

Response conflict with Simon interference was present in *incongruent* trials with a *compatible* SR-mapping and in *congruent* trials with an *incompatible* SR-mapping, supported by RTs, errors, and contralateral N300. This initially complex finding reflects the fact that with an incompatible SR-mapping, locations that are congruent with the target arrow are incongruent with the correct response. Crucially, measures of location-related response conflict in performance and motor activation were not eliminated with the reversed mapping. This supports the assumption that Simon interference represents automatic response activation via the direct route, and is unaffected by the task instruction, which we assume to be processed via the indirect route. It is also important to note that measures of response conflict in errors and motor preparation with Simon interference appear to be of greater magnitude in the current study than in our earlier comparison (Mansfield et al., 2013). We suspect that this difference largely reflects the unpredictability of Simon interference in the current mixed-interference design, which implies that resolving interference will have to rely more upon online control and less upon preparatory measures, but to some extent the inclusion of neutral flankers with Simon interference in the current study might have increased the magnitude of Simon effects.

3.5.3 Response Conflict with Eriksen Interference

First we confirmed that Eriksen interference was effective at the response level; in compatible blocks, performance with neutral trials was similar to performance with congruent flankers and significantly better than performance with incongruent flankers. Consequently, we assume that interference by incongruent flankers reflects mainly S-R interference. As expected, both contralateral N250 and contralateral N300 were reduced with *incongruent* Eriksen interference in compatible blocks, confirming flanker-related response conflict in both of these intervals. The N250 interval is in line with the temporal dynamics of Eriksen interference in our previous study, but Mansfield et al. (2013) also found support in a later interval in L-ERPs (N350) that Eriksen interference was resolved, reflected by enhanced contralateral negativity with incongruent flankers. Visual inspection of contralateral L-ERPs in the current experiment (figure 3.3) suggests the presence of a similar component with Eriksen interference, but this component was not as distinct as N350 in Mansfield et al., nor did it demonstrate enhanced negativity compared to congruent Eriksen interference. We assume that mixing interference in the current study delayed the

temporal dynamics of flanker-related response conflict and its resolution, as well as increasing response conflict with Simon interference, due to the increased number of alternative response strategies needed.

What is particularly interesting is that with an incompatible mapping Eriksen interference effects disappeared in both error and motor preparation. We had predicted that in incompatible blocks Eriksen congruency effects would be similar but slightly reduced; this is because translating the arrow direction might be expected to take longer, but incongruent flankers should activate the incorrect response via the indirect route, even with the reversed mapping. This prediction fits with our RT results, but cannot account for the elimination of Eriksen effects in errors and motor preparation. There are at least two logical accounts for this elimination: the first account is that with an incompatible SR-mapping incongruent flankers activated both the incorrect response via the indirect route and the correct response via the direct route to the extent that these effects cancelled each other out; a second account is that participants applied a different strategy in incompatible blocks, such as increased attention on the central target and/or delaying response execution to allow additional time to translate the target. The rest of the discussion integrates all performance and ERP measures in order to identify the most likely accounts for conflict resolution with Simon and Eriksen interference.

3.5.4 Support for Domain-Specific Mechanisms of Control

The clearest result from analyses of midline ERPs is that online cognitive control (reflected at N1 and N2) was enhanced for all Simon stimuli relative to all Eriksen stimuli. At first sight, this finding supports the hypothesis that resolution of Simon interference, incurred as direct automatic response activation by irrelevant stimulus locations, can be resolved by a voluntary suppression of the direct route (e.g. Kornblum et al., 1990). However, neither N1 nor N2 were sensitive to SR-mapping or Congruency, nor were they associated with the conditions that demonstrated the greatest conflict in performance measures. Together these findings imply that N2 in this task did not reflect detection of response conflict, but instead suggests that online control can be influenced by fast detection of salient stimulus attributes. Potentially in the current task, N1 reflects additional visual discrimination processing when the target is not at fixation (c.f. Vogel & Luck, 2000), and N2 reflects subsequent inhibition of the direct route in *anticipation* of location-related response conflict.

The idea that accounting for adjustments to cognitive control does not require detection of response conflict has been proposed before (Scherbaum et al., 2012), and the current finding appears to support direct control adjustments in response to stimulus features alone, but further research is needed to control for other cognitive processes reflected in the amplitude of N1 and N2, which we will discuss below.

So far, there is support for increased online control with unilateral stimuli, but it is less clear how this control mechanism might differ to the cognitive control involved in resolving Eriksen interference. Our hypothesis was that Eriksen interference might be resolved by increased attention to the target (with an associated inhibition of flanker processing), and potentially a general suppression of response activation (c.f. Band & van Boxtel, 1999) to allow sufficient time to identify the target and associated response. With a blocked interference design (Mansfield et al., 2013), we found evidence of response conflict at contralateral N250, and a late midline/contralateral component (N350) in Eriksen task blocks that was enhanced with incongruent flankers, suggesting control processes involved in the resolution of conflict in that interval. But in the current mixed interference experiment, with a compatible mapping there was still evidence of Eriksen response conflict (reduced negativity) at contralateral N300, and no visible suggestion of conflict resolution (enhanced negativity) for incongruent flankers at a later interval. Assuming that enhanced negativity at midline frontal N2 reflects increased cognitive control (c.f. Folstein & van Petten, 2008), then it seems that there was little or no increase in online cognitive control during resolution of Eriksen interference with our mixed interference design. However, this finding does not rule out the possibility that in all mixed interference blocks, participants prepared to deal with Eriksen interference and adjusted their response strategy upon detection of Simon stimuli.

In favor of the idea of a preparatory bias toward resolving Eriksen interference, Eriksen effects were reduced and even eliminated with the reversed mapping, which could be interpreted as a further increase in attention to the task goals (translating the target arrow and ignoring the flankers) in incompatible blocks. In support of this interpretation, in incompatible blocks responses were generally slower but there was no SR-Mapping effect on errors, even in comparisons of neutral trials. This suggests that participants were slightly more cautious with a reversed SR-mapping, which could reflect control over the baseline level of response activation (c.f. Bogasz et al., 2010). As such, if our results support domain

specific mechanisms of control, then resolving interference incurred by direct SR-priming, as with Simon interference, likely involves voluntary suppression of the direct route, whereas resolving interference incurred via the indirect route, such as with Eriksen interference, likely involves increased preparatory control over task goals in the indirect route.

3.5.5 Alternative Accounts

There are other potential interpretations of our results that should be considered, which could shed doubt on the hypothesis of independent mechanisms of conflict resolution for each type of interference. For example, both the N1 and N2 enhancements for Simon stimuli might reflect the slightly lower frequency of unilateral (40%) compared to centrally-presented (60%) stimuli in each block. This is in line with research on the auditory oddball N1 and mismatch negativity (see Näätänen et al., 2007, for a review), and the finding that N2 is enhanced with low frequency events (e.g. Nieuwenhuis et al., 2003). 40% is not considerably low frequency, but perhaps just low enough to demonstrate stimulus probability effects on both N1 and N2. Another possibility is that unilateral stimuli generally elicit greater negativity, which has been suggested by Luck et al. (1990), who compared unilateral to bilateral stimuli in the 75-250ms post-stimulus range. There was no suggestion of increased negativity with unilateral stimuli in the study by Mansfield et al. (2013), but the blocked design in that study might have meant that increased negativity with unilateral stimuli was disguised by the relatively lower frequency of centrally-presented (neutral) stimuli.

The elimination of Eriksen interference in errors and motor preparation in incompatible blocks could also be accounted for by a traditional continuous flow account of Eriksen effects (e.g. Eriksen & Schultz, 1979), such that flanker stimuli automatically activate the corresponding response, and then presumably via the direct route. In favor of this account, centrally presented arrow stimuli have been reported to produce small but reliable Simon effects (Cespón et al., 2013; Eimer, 1995), but to be limited to slower RTs (Cespón et al., 2013). This finding is especially relevant because usually distributional analyses of traditional Simon effects reveal interference effects to be largest for shorter RTs, implying that interference from incongruent locations depends upon temporal overlap between selection of the relevant and the irrelevant stimulus dimension (de Jong et al., 1994, but see also Zhang & Konblum, 1997). Indeed, the more recent study by Cespón and colleagues used distributional analysis to compare Simon interference by location vs. arrow direction (color

was the response-relevant stimulus dimension), demonstrating that location interference had earlier effects compared to arrow-direction interference. The difference in the timing of location vs. arrow Simon effects in the study by Cespón et al. (2013) presumably reflects the additional time required to identify an arrow compared to a location. As such, interference via the direct route would be later for flanker arrows than for locations, implying that the same mechanisms of control could be applied to resolve both types of interference, but with different temporal dynamics.

If Eriksen interference is incurred via the direct route, then with a reversed SR-mapping, incongruent flankers would automatically activate the same response as the central target arrow, but congruent flankers would activate the opposite response, implying that performance should be better with incongruent flankers than with congruent flankers. While our results suggest that this is completely the case for Simon interference, it is not the case for Eriksen interference, because at least in RTs Eriksen effects were not eliminated by reversing the mapping instruction. However, we can't rule out the possibility that Eriksen interference is incurred via both routes of a dual-route model, even if such effects reflect predominantly the indirect route. This implies that with incongruent flankers in incompatible blocks, direct-route effects and indirect route effects might cancel each other out, resulting in an elimination of Eriksen effects similar to our findings in errors and RTs.

3.5.6 Consolidating the Evidence

While we cannot completely rule out the possibility that midline components might have partially reflected stimulus probability, and that reversing the SR-mapping might have automatically reduced Eriksen effects, we do interpret the results as support that Eriksen and Simon interference reflect different sources of response conflict and to some extent independent mechanisms of control. Presenting both types of interference in the same experimental blocks allowed us to disentangle these mechanisms. We expect that participants must have prepared for the most difficult source of interference (Eriksen, as reflected in compatible blocks both in the current study and in Mansfield et al., 2013), and updated response strategies accordingly upon detection of unilateral stimuli. As such, resolving Eriksen interference relied upon preparatory control over processing the central target via the indirect route, and resolving Simon interference relied upon online control involved in detecting salient stimulus attributes and suppressing the direct route. This

account seems plausible if you consider that all stimulus types included flankers that needed to be ignored, even neutral and Simon stimuli. Potentially, neutral trials were processed in a similar way to Simon trials due to the presence of squared flankers, for which we found some support in the amplitude of midline N2.

There is some suggestion that the bias toward reducing Eriksen interference might have been stronger with the reversed SR-mapping. For example, the dissociation between RTs and errors (even for neutral trials), such that responses were generally slower but there was no comparable increase in errors, suggests that participants were slightly more cautious in incompatible blocks. However, there were no SR-mapping effects on midline N1 or N2, and the only SR-mapping difference in motor preparation was the elimination of Eriksen response conflict. If participants were strategically delaying response execution by reducing the level of response activation (c.f. Band & van Boxtel, 1999), then we might have expected a similar elimination of Simon interference in incompatible blocks. In sum, it seems most likely that any increased control in incompatible blocks was only beneficial to reducing Eriksen interference via the indirect route, offering additional support for the idea that these types of interference are resolved by independent mechanisms.

Such a strategic account fits with the Dual Mechanisms of Control (DMC) model (Braver et al., 2007), which differentiates between proactive and reactive cognitive control. In the current experiment, proactive control might be involved in maintaining the multiple SR-bindings in working memory, focusing on the target, ignoring the flankers, and potentially delaying response activation to allow sufficient time for decision processes to identify the correct response (c.f. Bogacz et al., 2010); reactive control might be involved in detecting (response) conflict or other salient stimulus attributes, correcting preferential response activation when it favors the incorrect response, and potentially suppressing direct response activation following detection of response conflict. As such, a strategy is a form of proactive control, but specific strategies might prepare for additional adjustments by reactive control when needed, such as in the case of detection of response conflict. In the current mixed interference design, we assume that Eriksen interference was resolved primarily by proactive control and Simon interference was resolved by a reactive suppression of the direct route, triggered by detection of salient stimulus attributes (e.g. square flanker stimuli). However, in line with the DMC, we assume that cognitive control is flexible and dynamic,

such that in certain circumstances direct route suppression might be part of the task goals maintained by proactive control.

3.5.7 Limitations and Future Directions

The most significant limitation of the current study is its sheer complexity, which posed an even greater challenge in interpreting the results than when making predictions in relation to a dual-route model. The predictions alone were in terms of three-way interactions between Congruency, Interference-Type, and SR-Mapping, and then in relation to response conflict predictions differed for activity contralateral or ipsilateral to the response hand. The complexity of the results is reflected in the dissociation between effects on RTs and accuracy, which often appeared to favor different accounts. Crucially, interpretations depended upon the extent to which flankers are assumed to incur automatic response activation via both the direct and indirect routes of a dual-route model, especially when considering the added complication of SR-mapping effects. The latter complexity could be addressed in the future by simulations comparing different magnitudes of interference via each route. Another issue that has not been resolved by the current study is the extent to which interference can be prevented by preparatory (proactive) control. Comparison with our earlier study with a blocked interference design (Mansfield et al., 2013) offered some insight into this aspect, but future studies with ERP measures could make a more direct comparison of control mechanisms with blocked and mixed interference.

3.5.8 Conclusions

We confirmed independent sources of response conflict for Simon and Eriksen interference in measures of performance and motor preparation by comparing the effect of reversing the SR-mapping on each type of interference. In line with a dual-route model, Simon interference is incurred by automatic response activation via the direct route and Eriksen interference is incurred mostly via the indirect route. We also found some support that these two types of interference are resolved by domain-specific control mechanisms that can be described in terms of proactive and reactive control (Braver et al., 2007). We suspect that Eriksen interference was mostly prevented by proactive control and Simon interference resolved by reactive control, in line with the finding of an enhanced N1 and N2 with all Simon stimuli. We interpret these results to reflect a strategy with mixed interference that

prepares for the worst (incongruent flankers), but upon detection of relevant stimulus features (reflected in N1) subsequently suppresses the direct route (reflected in N2). This latter finding suggests that control adjustments do not need to depend upon detection of response conflict, but instead can use detection of salient stimulus attributes to trigger control adjustments (c.f. Scherbaum et al., 2012).

Chapter 4:
Proactive and reactive control in S-R compatibility:
A brain potential analysis

We investigated how proactive and reactive control facilitates performance in mixed stimulus-response compatibility (SRC) tasks. SRC effects were eliminated in mixed tasks, and reversed following incompatible trials. In mixed tasks, early preferential response activation was present in stimulus-locked LRPs, but reduced following incompatible trials. In ERPs, stimulus-locked N2 was enhanced in all mixed trials, but was not significantly influenced by the preceding trial. A response-locked fronto-central negative component (N-120), peaking just before the response, was largest for mixed compatible trials preceded by incompatible trials. This N-120 was paired with an enhancement to the peak of the response-locked LRP. Proactive control is involved in selection of an S-R mapping via the indirect route of a dual-route model. Reactive control corrects the S-R mapping, particularly when alternating between S-R mappings.

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4.1 INTRODUCTION

Research in cognitive neuroscience continues to reveal how dynamic and flexible the human brain is. While many actions can be performed quite automatically, human beings can also achieve complicated goal-directed behavior (e.g., James, 1890; Shiffrin & Schneider, 1977). Such accurate performance is enabled by cognitive control, which delegates tasks and resolves interference between possible responses (e.g. Norman & Shallice, 1986). A recent concept of cognitive control differentiates between dual-mechanisms (Braver, Gray, & Burgess, 2007; Forstmann, Ridderinkhof, Kaiser, & Bledowski, 2007). While proactive control exhibits goal-relevant early selection, reactive control is involved in specific late correction. Here we will use electrophysiological measurements to investigate the temporal dynamics of how proactive and reactive cognitive control overrules automatic processes, using a stimulus-response compatibility (SRC) task.

SRC effects are typically tested in blocked tasks, in which a spatially compatible S-R mapping (left-stimulus signals left-response) is faster and incurs less errors than an incompatible S-R mapping (left-stimulus signals right-response) (Fitts & Deininger, 1954). This phenomenon is generally referred to as the "mapping effect" (Kornblum, Hasbroucq, & Osman, 1990). Dual-route models have been proposed to account for the mapping effect (De Jong, Liang, & Lauber, 1994; De Jong, 1995; Kornblum et al., 1990; Ridderinkhof, van der Molen, & Bashore, 1995; Stoffels, 1996a; van Duren & Sanders, 1988). According to these models, the mapping effect is a result of the difference in performance via a direct and an indirect route. Compatible trials will benefit from S-R priming via the direct route, and will also involve more efficient stimulus-response selection via the indirect route.

In blocked tasks, the unique S-R mapping benefits from practice. Although S-R priming via the direct route maintains the mapping effect, via the indirect route the task becomes increasingly automatic (c.f. James, 1890; Posner & Snyder, 1975), which can decrease the mapping effect. In mixed tasks that include both compatible and incompatible S-R mappings, mapping effects are greatly reduced (Christensen, Ivkovich, & Drake, 2001; Heister & Schroeder-Heister, 1994; Proctor & Vu, 2002; De Jong, 1995; Shaffer, 1965; Stoffels, 1996a; Stoffels 1996b; van Duren & Sanders, 1988; Vu & Proctor, 2004) and sometimes even reversed (Jennings, van der Molen, van der Veen, & Debski, 2002). The reduction of the mapping effect primarily reflects a disadvantage to mixed compatible trials,

while mixed incompatible trials are comparatively unaffected (e.g. Vu & Proctor, 2004). Current interpretations of performance in mixed S-R mapping tasks assume that both compatible and incompatible trials are processed via the indirect route, and that the direct route is suppressed (De Jong, 1995; Vu & Proctor, 2004). However, eliminating the ambiguity of the S-R mapping, by presenting cues in advance, can restore the mapping effect, but only when the cue-target interval is sufficiently long (De Jong, 1995; Jennings et al., 2002; Stoffels, 1996b).

Performance in interference tasks is also especially dependent upon sequential trial effects, whereby SRC effects are maintained following compatible trials and eliminated following incompatible trials (Burle, Allain, Vidal, & Hasbroucq, 2005; Hommel, Proctor, & Vu, 2004; Jentzsch & Leuthold, 2005; Praamstra, Kleine, & Schnitzler, 1999; Stürmer, Leuthold, Soetens, Schröter, & Sommer, 2002; Wendt, Kluwe, & Peters, 2006). This reduction of SRC effects has often been interpreted as suppression of S-R priming following incompatible trials (Duncan, 1978; De Jong, 1995; Kornblum et al., 1990; Praamstra et al., 1999; Ridderinkhof, 2002; Shaffer, 1965; Stoffels, 1996b; Stürmer, et al., 2002, Stürmer, Redlich, Irlbacher, & Brandt, 2007; Vu & Proctor, 2004). Suppression of S-R priming might represent a form of cognitive control involved in the reduction of S-R mapping effects. Recent research has demonstrated how sequential dependencies involving several preceding trials reflect task level effects (Lindsen & De Jong, 2010), suggesting that control strategies are also active between trials.

A few studies have investigated sequential S-R mapping effects in mixed tasks (De Jong, 1995; Jennings, et al., 2002; Stoffels, 1996a, 1996b). Similar to the influence of mixed compared to blocked mappings, the diminished S-R mapping effect (following an incompatible trial) reveals itself as a substantial delay to compatible trials. Jennings and colleagues (2002) interpreted the reversal of the S-R mapping effect in the mixed task as a bias towards the incompatible mapping, whereby application of an S-R mapping can be seen as application of a task schema (c.f. Norman & Shallice, 1986). Jennings and colleagues assumed that in mixed tasks the task schema for the incompatible mapping was most active, but that this task schema was voluntarily suppressed when the compatible mapping was required.

The task schema account of mixed SRC tasks is also comparable to the idea of “logical recoding” (Hedge & Marsh, 1975), which recodes the stimulus into the appropriate response

according to task requirements (e.g., left = right). The dual-process model of de Jong, Liang, and Lauber (1994) is based upon logical recoding. This model assumes that not only direct (unconditional) S-R priming is automatic in nature, but that indirect (conditional) S-R processing is also initially automatic. Therefore, with a logical recoding account, the S-R mapping that is most active will be applied automatically (conditional priming), until control processes are able to influence correct response selection. In other words, participants might automatically apply the incompatible mapping, and use additional control processes to overcome this recoding when necessary.

In terms of the cognitive control involved in the elimination or reversal of S-R mapping effects in mixed tasks, p*roactive* control might influence activation/suppression of a task schema (or logical recoding). Subsequently, *reactive* control might reactivate the appropriate mapping when this has been incorrectly applied by proactive control. The response conflict hypothesis assumes that cognitive control relies upon conflict-monitoring (Carter & van Veen, 2007; Botvinick, Braver, Carter, Barch, & Cohen, 2001; Yeung, Botvinick, & Cohen, 2004), involving a feedback-loop via the anterior cingulate cortex (ACC) and the dorsolateral prefrontal cortex (DLPFC). Conflict is usually assessed by means of simulation (Carter & van Veen, 2007; Botvinick, Braver, Carter, Barch, & Cohen, 2001; Yeung, Botvinick, & Cohen, 2004), but some studies have used electrophysiological measures to assess conflict (e.g. Masaki, Falkenstein, Stürmer, Pinkpank & Sommer, 2007; Bartholow, Pearson, Dikter, Sher, Fabiani, & Gratton, 2005). In terms of conflict, we expect that *proactive* control uses prior knowledge concerning the conflict expected in the task globally, while *reactive* control is related to the amount of conflict within specific trials. However, the focus of this study is cognitive control rather than conflict, and its (in)sensitivity to trial-to-trial effects. As such we assume that *proactive* control will be sensitive only to task effects, while *reactive* control will be sensitive to within and between-trial effects (i.e., the S-R mapping in the current and preceding trial).

A successful means of investigating S-R processing in interference tasks is the lateralized readiness potential (LRP) (De Jong et al., 1994; Eimer, 1995; Gratton, Coles, Sirevaag, Eriksen, & Donchin, 1988; Stürmer & Leuthold, 2003). The LRP is a difference wave that compares the relative amount of contra- and ipsilateral motor cortex activation in left- versus right-hand responses, representing the build-up of response activation in terms of correct and incorrect response channels. On incompatible trials, prior to the onset of the

final LRP an early deflection towards the incorrect response (the "Gratton dip", Gratton et al, 1988) has been detected in many interference tasks (De Jong et al., 1994; Gratton et al., 1988; Vallesi, Mapelli, Schiff, Amodio, & Umiltà, 2005; van der Lubbe, Jaśkowski, Wauschkuhn, & Verleger, 2001). By testing the direction (correct vs. incorrect) and timing of such preferential response activation in LRPs, we can assess the extent to which interference reflects early S-R priming via the direct route, and/or later conditional effects via the indirect route. De Jong and colleagues (1994) reported finding both direct unconditional and indirect conditional response activation in LRPs with an S-R mapping task. However, the authors did not investigate the role of sequential effects, and could not conclude whether or not suppression of S-R priming was involved. If S-R priming is suppressed in the blocked incompatible task, then we can expect LRP onset to be delayed without the presence of a Gratton dip. However, efficient processing via the indirect route can also explain the absence of a Gratton dip, without assuming suppression of S-R priming. If S-R priming via the direct route is suppressed in mixed tasks following incompatible trials, then we can expect to find a reduction of S-R priming for these trials *early* in the LRP. But again, a logical recoding account of mixed tasks can also explain the absence of a Gratton dip: if the S-R mapping of the preceding trial is initially applied in the current trial via the indirect route, this will also resemble a reduction in S-R priming following incompatible trials. On the other hand, if the S-R mapping of the preceding trial is activated later on in stimulus-response translation, then early S-R priming (or its absence) will be followed by a late redirection in the LRP. We assume the blocked tasks as a baseline, whereby effects that are incurred in mixed tasks later than the LRP onset in blocked tasks can best be interpreted as indirect route effects.

We will investigate *proactive* control by analyzing the N2 component, a stimulus-locked ERP component that has previously been linked to cognitive control, peaking at fronto-central sites around 200 – 360 ms after stimulus presentation. Assuming that stimulus-locked N2 represents *proactive* control, then we expect it to be enhanced by mixed tasks, but not specifically by within or between-trial effects. Whether the control measured at N2 in interference tasks reflects response inhibition (Carriero, Zalla, Budai, & Battaglini, 2007; Falkenstein, Hoormann, & Hohnsbein, 1999; Gajewski, Stoerig, & Falkenstein, 2008; Heil, Osman, Wiegelmann, Rolke, & Henninghausen, 2000; Kopp, Mattler, Goertz, & Rist, 1996; Kopp, Rist, & Mattler, 1996; van Boxtel et al., 2001), or evaluative processes involved in the detection of conflict (Carter & van Veen, 2007; Nieuwenhuis et al., 2003; van Veen &

Carter, 2002; Yeung et al., 2004), is still a discussion point. For the purposes of this study, stimulus-locked N2 can be interpreted as an ERP component manifesting cognitive control, and the nature of such control will be revealed by its (in)sensitivity to particular experimental conditions.

Unlike *proactive* control, we assume that *reactive* cognitive control *will* be sensitive to within and between-trial effects. In many interference tasks, increased conflict is expected in incompatible trials. However, in a mixed S-R mapping task, increased conflict might be expected on compatible trials (in the case of a bias towards the incompatible mapping), and when alternating between S-R mappings. Furthermore, we assume that *reactive* control will be time-locked to the response. Yeung and colleagues (2004) presented response-locked waveforms in which an enhanced fronto-central negativity was visible 90 ms prior to the response on incongruent trials. They assumed this response-locked component to represent the same mechanisms as their stimulus-locked N2 (344 ms). This assumption fits with the mean RTs for incongruent trials in their study (421 ms) because the sum of the timing of the two components roughly equals the RT. However, we expect that in the current mixed S-R mapping tasks RTs will be much longer, and there will be less temporal overlap between response-locked and stimulus-locked components. The response-locked control component should be both temporally and functionally differentiable from stimulus-locked N2, revealing separate underlying control mechanisms. We predict that particularly mixed compatible trials preceded by incompatible trials will involve the most interference, resulting in most errors and an enhanced fronto-central negativity (representing *reactive* control) just prior to the response.

On trials in which an error is committed, a large fronto-central Error Negativity (N_e) or Error Related Negativity (ERN) is produced (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1991; Gehring, Goss, Coles, Meyer, & Donchin, 1993), immediately following the response. However, on correct trials a negative component with a similar scalp distribution to the ERN can be detected. This component has been referred to as the Correct Related Negativity (CRN) or Correct Negativity (N_c), and is smaller in amplitude compared to the ERN (Falkenstein, M., 2002; Masaki, Falkenstein, Stürmer, Pinkpank, & Sommer, 2007; Vidal, Hasbroucq, Grapperon, & Bonnet, 2000). Similar to Vidal and colleagues (2000), we assume that CRN (and ERN), reflects a reactive comparison process concerning the expected and the

actual responses activated. We expect that CRN will also be sensitive to the trials that result in most errors.

In sum, we expect to find increased measures of *proactive* and *reactive* cognitive control in mixed tasks, as outlined in Table 4.1 in terms of a dual-route model. We assume that in mixed tasks the S-R mapping is initially activated by *proactive* control via the indirect route of a dual-route model, influenced by the S-R mapping in the preceding trial and with a bias towards the incompatible mapping. We expect an enhanced N2 in mixed tasks that is insensitive to the S-R mapping in the current and/or preceding trial. We assume that *reactive* control will subsequently activate the correct S-R mapping when this is incorrectly applied by *proactive* control. We therefore expect an enhanced fronto-central negativity just prior to the response, and an enhanced CRN following the response, for mixed compatible trials and mapping alternations. We will also test the hypothesis that S-R priming via the direct route is suppressed in mixed tasks, and specifically in trials preceded by the incompatible mapping. If this is true, then we can expect preferential response activation in mixed tasks to be reduced or delayed from early on in LRPs, particularly following incompatible trials.

Hypotheses regarding performance in mixed blocks	**Predicted effects on behavior**	**Predicted effects on LRPs**	**Predicted effects on control components**
Proactive control (early S-R mapping selection via indirect route).	General delay to mixed tasks. Eliminated mapping effects.	Delayed LRP. Reduced S-R Mapping effects.	Enhanced N2 in all mixed trials.
Reactive control (late S-R mapping selection via indirect route).	Delay to mixed tasks, particularly on S-R mapping alternations.	Late redirection in stimulus-locked LRPs.	Response-locked fronto-central negativity prior to the response.
Suppression of direct route (proactive control) following incompatible trials.	Eliminated mapping effect in mixed tasks, particularly following incompatible trials.	Delayed LRP. Reduced S-R Mapping effects (early on in LRPs) following incompatible trials.	Enhanced N2 (proactive control),particularly following incompatible trials.
Bias towards incompatible mapping in the indirect route (proactive), with late selection of the compatible mapping (reactive).	Reversed mapping effect, particularly following incompatible trials.	Delay and late redirection in LRP for mixed compatible trials, particularly following incompatible trials.	Enhanced N2 (proactive control). Response-locked fronto-central negativity prior to the response (reactive control) in mixed compatible trials.

Table 4.1. Major hypotheses tested in the current study, in line with a dual-route model of stimulus-response compatibility. Predictions concerning proactive and reactive control are outlined for behavior, LRPs, and ERPs.

4.2 METHODS

4.1.1 Participants

18 first-year psychology students took part in the study in exchange for course credit. Two participants were excluded from analyses due to excessive error rates, and two due to excessive EEG artifacts. The remaining 14 participants (10 men and 4 women) were all right-handed, with ages ranging from 19 to 37 years (mean 22 years). All were neurologically healthy and had normal or corrected-to-normal vision.

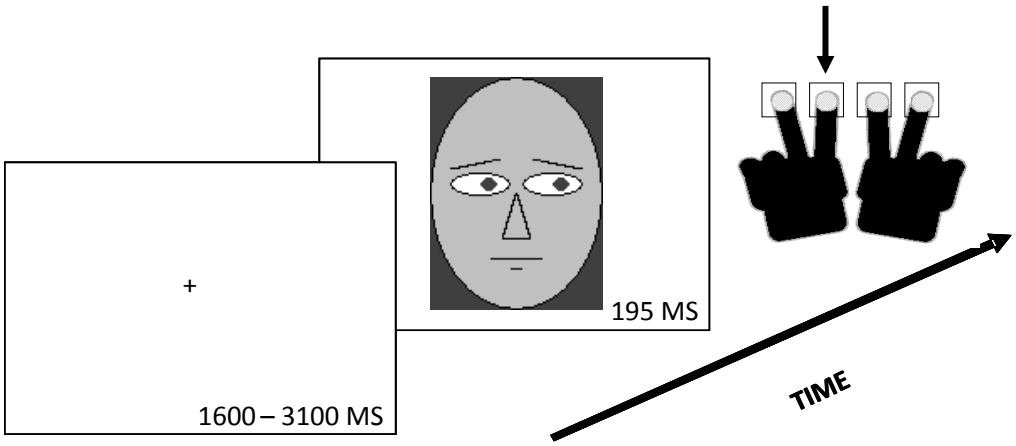

Figure 4.1. Example of an incompatible mapping trial with the narrow gaze eccentricity.

4.1.2 Stimuli and Apparatus

The stimuli (see Figure 4.1) were schematic faces, with a height of 3.6 cm and width of 2.7 cm (2.2 visual degrees), a light skin color, and blue eyes. The gaze stimuli were centered on a dark gray background. The eyes (each 1 cm wide, 0.8 degrees, with 2 mm space between the eyes) had four possible gaze directions: wide left, wide right, narrow left, and narrow right. For the wide eccentricity stimuli, the colored pupil was shifted 3.2mm (0.3 degrees) to the left/right of the center of each eye; for the narrow eccentricity stimuli, the colored pupil was shifted 1.2mm (0.1 degrees) to the left/right of the center of each eye. Similar to the design by Stoffels (1996b), in the current study the factor Eccentricity cues the S-R mapping. This factor was selected because it enables the S-R mapping to be cued simultaneously to target presentation. A good alternative to horizontally displaced stimuli is gaze direction stimuli, which have previously demonstrated an S-R mapping effect (Ansorge, 2003; van den Wildenberg & van der Molen, 2004). Importantly, gaze stimuli can be presented at fixation,

limiting visual asymmetries (see Praamstra, 2007) and unwanted horizontal eye-movements. Participants were seated in a dimly lit, sound-attenuated, electrically shielded room, and a 14-inch monitor was positioned at eye-level at a distance of 70 centimeters.

4.1.3 Tasks and Procedure

Participants were required to respond as quickly as possible, using index and middle fingers, by pressing one of four designated response buttons. Response fingers and buttons were positioned spatially equivalent (from left to right) to the orientation of the gaze stimuli. All participants took part in four choice response tasks; two of which with blocked S-R mappings and two with mixed S-R mappings. In the blocked compatible task, participants were required to press the button that corresponded spatially to the gaze direction of the stimuli (compatible mapping). In the blocked incompatible task, participants had to press the button that was spatially opposite to the gaze direction of the stimuli (incompatible mapping). In one of the mixed tasks, participants had to apply the compatible mapping to the wide eccentricity stimuli and the incompatible mapping to the narrow eccentricity stimuli. In the other mixed task, the combination of wide/narrow eccentricity with compatible/incompatible mapping was reversed. The order in which the four tasks were tested was counterbalanced for the first sixteen participants, and the last two participants repeated the task order of participants already excluded from analyses. Each task consisted of two measurement blocks of 180 trials (approximately 10 minutes each), which were always tested consecutively. Prior to the test phase there was a training session, during which all tasks were practiced consecutively in 4 short blocks of 40 trials in the following order: blocked compatible, blocked incompatible, mixed with the incompatible mapping applied to the narrow eccentricity stimuli, then mixed with the incompatible mapping applied to the wide eccentricity stimuli. The standard order of practice trials was chosen due to the complexity of the mixed tasks. A fixation cross (+) was present on the screen, both before and after stimulus presentation. Each trial began with a variable inter-trial interval, between 1600 ms and 3100 ms (in steps of 1 ms), followed by the presentation of the gaze stimulus for 195 ms. The inter-trial interval was initiated as soon as the participant responded.

4.1.4 EEG Recording and Analysis

EEG was recorded from 47 locations using active sintered electrodes (BioSemi Active 2) mounted in an elastic cap, with a sample rate of 256 Hz. Vertical eye movements were recorded from electrodes positioned above and below the right eye, and horizontal eye movements from electrodes at the outer canthus of each eye. Data analysis was performed on ERPs using the Brain Vision Analyzer software package. Raw data were referenced off-line to average mastoids and all channels were band-pass filtered from 0.1-12 Hz. The filtered data were segmented into epochs ranging from 300 ms prior to the stimulus until 1 second after the stimulus, and coded according to condition, accuracy, and outliers. Segments were visually inspected and segments with large artifacts (3% of all trials) were removed manually prior to EOG correction. Blinks (in 38% of segments) were detected and corrected using the regression technique by Gratton and Coles (1983). Horizontal eye movements were calculated as "right – left", and this difference wave was subjected to the same automatic rejection procedure as the remaining EEG channels, which rejected every segment that had a voltage step larger than 100 µV, or an amplitude range of more than 120 µV, on any channel (5% of all trials). Baseline correction was based on the 100 ms prior to stimulus presentation, for both stimulus- and response-locked waveforms. Epochs were also segmented time-locked to the response, ranging from 700 ms prior until 200 ms after the response. Finally, stimulus- and response-locked ERPs were averaged for each participant per condition, excluding errors and outliers. Each of the conditions in the main analyses (Task x S-R Mapping) was represented by a maximum of 360 and a minimum of 92 trials per participant. Each of the conditions in the sequential effects analyses of the mixed tasks (Previous S-R Mapping x Current S-R Mapping) was represented by a maximum of 180 and a minimum of 40 trials per participant.

L-ERPs were calculated according to the LRP formula incorporated in Brain Vision Analyzer: LRP = [C4(left-hand)-C3(left-hand)+C3(right-hand)-C4(right-hand)] / 2. Correct-side activation (contra-lateral to the correct response hand) was therefore represented by negative deflections and incorrect-side activation by positive deflections. This calculation was performed for electrode pairs C4/C3, PO8/PO7, T8/T7, FC2/FC1, and horizontal eye movements (Figure 4.3). Asymmetries and differences between conditions were largest at C3/C4 (Figure 4.4, lower panel), upon which all LRP analyses have been

performed. Paired t-tests against zero were performed on LRP amplitudes at every time-sample following stimulus onset (Figure 4.5). LRP onsets were defined as the first sample in a long interval (≥140 ms) of significant correct response activation (c.f. Smid, Mulder, & Mulder, 1987 & 1990). In mixed tasks, prior to LRP onset, early deflections of brief preferential response activation were expected in an interval later than LRP onset in the blocked compatible condition but earlier than LRP onset in the relevant mixed condition. Assessment of significant intervals of brief early preferential response activation was based on the method of Guthrie and Buchwald (1991) for testing difference waves ($t(13)>2.16$, $p<.05$), assuming a high autocorrelation ($ø = 0.9$). In order to precisely detect the temporal interval of mapping effects in LRPs, new difference waves were calculated comparing LRPs in compatible and incompatible trials (per Task, and in the mixed tasks according to the S-R mapping in the preceding trial). These difference waves were subjected to permutation tests at each sample (using the method of Blair & Karniski, 1993) to precisely determine the intervals in which the mapping effect was significant in LRPs (to a 95% confidence interval). Response-locked LRPs were calculated for the mixed tasks only, from which an area around the peak of the LRP was selected (from -160 ms until -20 ms), and the average amplitude in this interval was exported for sequential effects analyses.

For the stimulus- and response-locked ERPs, a CSD transformation (Perrin, Pernier, Bertrand, & Echallier, 1989, 1990), with order of splines 3, and maximum degree of polynomials 15, was performed on the participant's condition means. This procedure transforms the surface EEG based on spherical splines, eliminating the need for an external or average reference (see also Ferree, 2006). CSD transformation helped to differentiate the fronto-central N2 and its effects at FCz, avoiding contamination by the simultaneous occurrence of motor activation. The peaks of the N2 were detected automatically at FCz only, where both amplitude and effects were maximal on inspection of grand averaged ERPs, between 250 – 375 ms after stimulus-onset.

In response-locked ERPs, a negative component was detected, peaking 120 ms prior to the response (Figure 4.6, top panel). This response-locked N-120 coincided with P3 and motor potential, appearing as a negative bump on a slow positive wave. However, applying a CSD transformation localized the component to Fz and FCz, and its effects to FCz. Three response-locked components were scored from the CSD trace at FCz, first for the main analyses of both tasks, and subsequently for sequential effects analyses of mixed tasks. The

peaks of N-120 were picked by hand (ranging from 199 to 82 ms prior to the response), as well as the positive peak prior to the response (from 160 to 23 ms prior to the response), and CRN (from 4 to 59 ms after the response). For one participant N-120 could not be detected, and N-120 and the positive component were both scored as the amplitude 121 ms prior to the response. N-120 and CRN were analyzed peak-to-peak, as the difference from the positive peak between them. This analysis does not exclude the possibility of experimental effects on the positive peak, but it was chosen to avoid the influence of baseline differences, and ensures a reliable comparison between the response-locked negative components.

A raster-like plot (EEGLAB, Delorme & Makeig, 2004) was constructed of stimulus-locked CSD-transformed amplitudes at FCz in mixed compatible trials for one representative participant (number 6). Participant 6 was selected as the response-locked N-120 component was also visible as a wide stimulus-locked component. All trials in the mixed compatible condition with the narrow eccentricity were stacked in order of increasing RT on the vertical axis, with time in ms on the horizontal axis. A color-scale displayed CSD-transformed amplitudes (blue = negative, red = positive) from – 219 to + 219 $\mu V/m^2$. This plot enabled an assessment of whether N-120 was a unique component that could be differentiated from stimulus-locked N2, and whether it was time-locked to the stimulus or to the response.

4.1.5 Statistical Analysis

Outliers (trials with an RT more than 2 SDs outside the participants' condition mean) and errors (5.4% of all data) were omitted from analysis of RTs, ERPs, and LRPs. In initial analyses of RTs including Response Hand, no effects of Response Hand were found. RTs and errors were analyzed by repeated-measures ANOVAs, including the factors Task (blocked, mixed), S-R Mapping (compatible, incompatible), and Eccentricity (wide, narrow). An additional RT analysis compared the two mixed tasks, to test for different effects of reversing the S-R Mapping according to Eccentricity, and included the factors Eccentricity-Mapping Combination (Wide=Compatible & Narrow=Incompatible, vs. Wide=Incompatible & Narrow=Compatible) and S-R Mapping. Initial ERP analyses included Eccentricity, but this factor did not interact with any other factors. Subsequent repeated-measures ANOVAs of ERPs included the factors Task and S-R Mapping. In sequential effects analyses of mixed-tasks, RT, error, ERP and response-locked LRP data were subjected to repeated-measures

ANOVAs accounting for Previous S-R Mapping and Current S-R Mapping. Stimulus-locked LRPs were analyzed by t-tests in order to detect specific intervals of motor preparation and early preferential response activation (see LRP recording and analysis for details).

4.2 RESULTS

4.2.1 Performance

Mean RTs, SEs and proportion of errors are summarized in Tables 4.2 and 4.3, and the relevant effects are illustrated in Figure 4.2. Excluding trials in which no response was given, errors were generally slightly faster than correct responses, in blocked tasks (correct 639 ms, incorrect 626 ms) and in mixed tasks (correct 742 ms, incorrect 726 ms). Mixed tasks (722 ms, 6% errors) suffered a general disadvantage compared to blocked tasks (618 ms, 3% errors), in RTs ($F(1,13) = 75.6$, $p < .001$, $\eta_p^2 = .853$), and in errors ($F(1,13) = 23.5$, $p < .001$, $\eta_p^2 = .644$). RTs were shorter with wide eccentricity stimuli than with narrow eccentricity stimuli ($F(1,13) = 14.8$, $p = .002$, $\eta_p^2 = .532$). An interaction in RTs between S-R Mapping and Eccentricity ($F(1,13) = 6.4$, $p = .025$, $\eta_p^2 = .331$) revealed that the S-R Mapping effect was larger for the wide eccentricity stimuli (51 ms, $F(1,13) = 26.7$, $p < .001$, $\eta_p^2 = .672$), than for the narrow eccentricity stimuli (11 ms, ns). Compatible responses (654 ms) generally yielded faster RTs than incompatible responses (685 ms), $F(1, 13) = 16.7$, $p = .001$, $\eta_p^2 = .562$. But most importantly, a highly significant interaction (Figure 4.2, left) between Task and S-R Mapping, revealed the S-R Mapping effect in mixed tasks to be eliminated in RTs ($F(1,13) = 30.0$, $p < .001$, $\eta_p^2 = .698$), and reversed in errors ($F(1,13) = 22.5$, $p < .001$, $\eta_p^2 = .634$). This interaction was confirmed by a strong standard S-R Mapping effect in RTs in blocked tasks only ($F(1,13) = 44.0$, $p < .001$, $\eta_p^2 = .772$), and a reversed S-R Mapping effect in errors in mixed tasks only ($F(1,13) = 21.2$, $p < .001$, $\eta_p^2 = .620$). Additional analyses of RTs in mixed tasks compared the two Eccentricity-Mapping combinations (each of the mixed tasks) on the S-R Mapping effect: An interaction between Eccentricity-Mapping Combination and S-R Mapping ($F(1,13) = 5.0$, $p = .044$, $\eta_p^2 = .277$) revealed that S-R Mapping effects were eliminated (ns) when the narrow stimuli required the incompatible mapping, and reversed (only as a trend; $F(1,13) = 3.6$, $p = .08$, $\eta_p^2 = .217$) when the wide stimuli required the incompatible mapping. As expected, the blocked versus mixed pattern in both reaction times and errors establish

the compatible mapping as paying the largest penalty when S-R mappings are mixed, while the incompatible mapping is relatively unaffected.

								Peak-to-peak	
Task	**S-R Mapping**	**Eccentricity**	**RT**	**SE**	**% Error**	**N2 amp**	**LRP onset**	**N-120**	**CRN**
Blocked	Compatible	Wide	555	15	1,9	-32	246	19	54
		Narrow	609	21	2,7	-37	246	16	49
	Incompatible	Wide	641	21	2,5	-31	320	14	52
		Narrow	667	24	4,5	-32	340	16	43
Mixed	Compatible	Wide	704	25	7,0	-39	414	24	66
		Narrow	750	21	7,8	-37	504	24	53
	Incompatible	Wide	720	20	4,4	-36	465	17	52
		Narrow	713	23	4,9	-40	414	17	49

Table 4.2. Mean RTs, SEs, percentage of errors, N2 amplitude, onset of response LRP, and N-120 and CRN peak-to-peak amplitudes, in both tasks.

								Peak-to-peak	
Prev. SRC	**Current SRC**	**Eccentricity**	**RT**	**SE**	**% Error**	**N2 amp**	**LRP onset**	**N-120**	**CRN**
Compatible	Compatible	Wide	660	23	2,7	-39	395	20	65
		Narrow	698	18	5,3	-35	516	23	56
	Incompatible	Wide	751	21	5,3	-37	488	23	55
		Narrow	740	25	5,5	-41	441	23	48
Incompatible	Compatible	Wide	751	29	10,5	-45	449	34	70
		Narrow	803	24	9,9	-38	520	32	50
	Incompatible	Wide	687	19	3,5	-37	492	16	51
		Narrow	683	22	4,2	-38	406	17	55

Table 4.3. Mean RTs, SEs, percentage of errors, N2 amplitude, onset of response LRP, N-120 and CRN peak-to-peak amplitudes, in mixed tasks, accounting for the S-R Mapping (SRC) in the previous (prev.) trial.

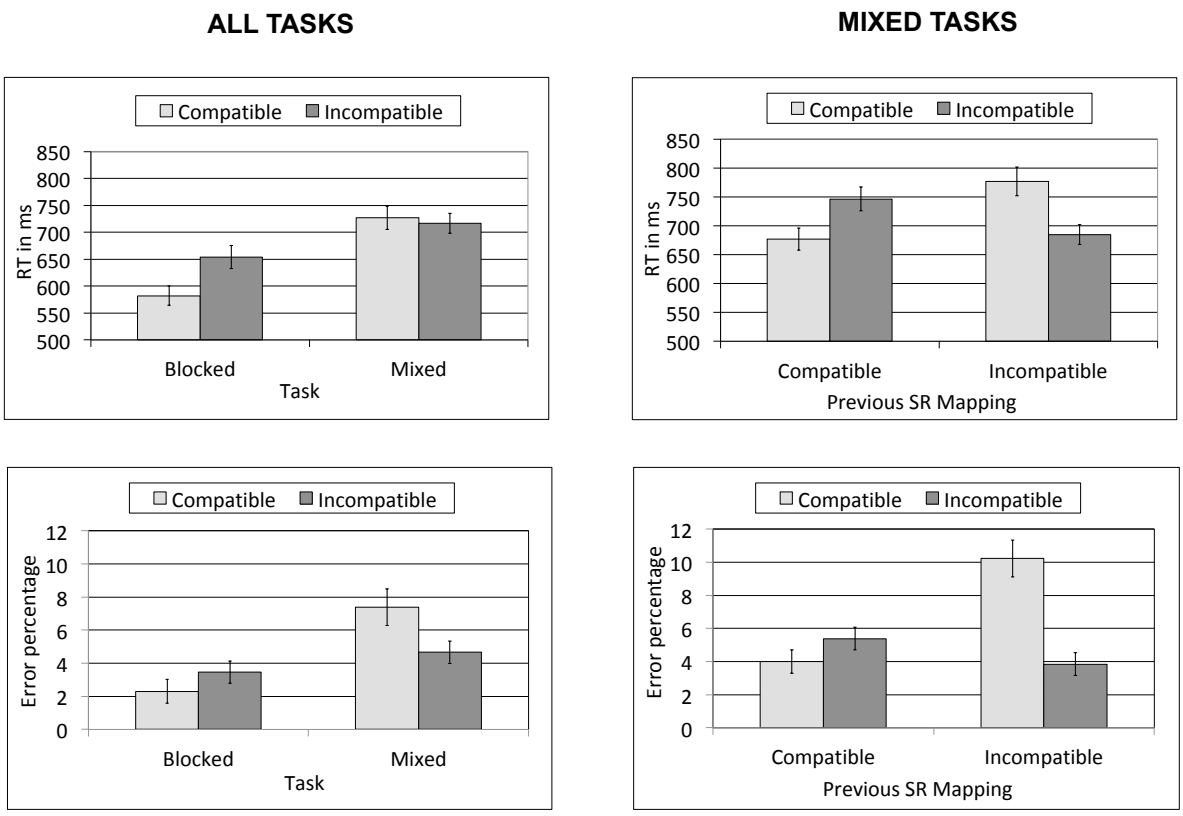

Figure 4.2. Left panel : mean RTs (top) and errors (bottom) in blocked and mixed tasks. Right panel: mean RTs (top) and errors (bottom) in mixed tasks, according to the S-R mapping in the previous trial.

In sequential effects analyses, a highly significant interaction (Figure 4.2, right) between Previous S-R Mapping and Current S-R Mapping (RTs, $F(1,13) = 98.6$, $p<.001$, $\eta_p^2 = .883$; Errors, $F(1,13) = 42.6$, $p<.001$, $\eta_p^2 = .766$) revealed the S-R Mapping effect following incompatible trials to be reversed rather than just eliminated, in RTs (-92 ms, $F(1,13)=42.4$, $p<.001$, $\eta_p^2 = .765$), and in errors ($F(1,13)=47.1$, $p<.001$, $\eta_p^2 = .784$). Following compatible trials, the S-R Mapping effect was maintained in RTs (67 ms, $F(1,13)=31.8$, $p<.001$, $\eta_p^2 = .710$), but absent in errors (1.5 % difference, ns). In order to further test the hypothesis of an advantage to the incompatible mapping in mixed tasks, post-hoc analyses were performed on trials involving a repetition compared to trials involving an alternation of S-R Mapping. When alternating between S-R Mappings, compatible trials (777 ms) were again slower than incompatible trials (746 ms), $F(1,13)=9.3$, $p = .009$, $\eta_p^2 = .418$.

4.2.2 Stimulus-locked L-ERPs

Grand averaged stimulus-locked L-ERPs, separated for Task, S-R Mapping and Eccentricity are illustrated in Figure 4.3. Prior to analyses grand averaged LRPs were collapsed over Eccentricity as this factor was intended only as an S-R Mapping cue that allowed four separate stimuli mapped to four separate responses in both tasks. In RT analyses Eccentricity interacted only with the current S-R Mapping (mapping effects were enhanced by wide eccentricity stimuli). Moreover, in the brain potential analyses, Eccentricity did not alter the focus of the current interest; i.e., the interactions between Task and S-R Mapping and between Previous and Current S-R Mapping (all *F*s < 4). Grand averaged LRPs at C4/C3 are depicted in Figure 4.4, and the results of the t-tests are depicted in Figure 4.5.

Final LRP onsets were defined as the initiation of significant correct response activation that continued for a long interval (c.f. Smid, Mulder, & Mulder, 1987 & 1990) of at least 39 consecutive samples (140 ms). In the blocked tasks, the LRP was quickly initiated and differed significantly from zero from 234 ms in compatible and from 324 ms in incompatible trials. In the mixed tasks, the final LRP commenced from 414 ms in compatible and from 434 ms in incompatible trials. When accounting for sequential effects in mixed tasks, trials that were preceded by compatible trials led to the LRP from 426 ms for compatible and 457 ms for incompatible trials. When preceded by incompatible trials, LRP onset attained significance from 469 ms for compatible and 426 ms for incompatible trials.

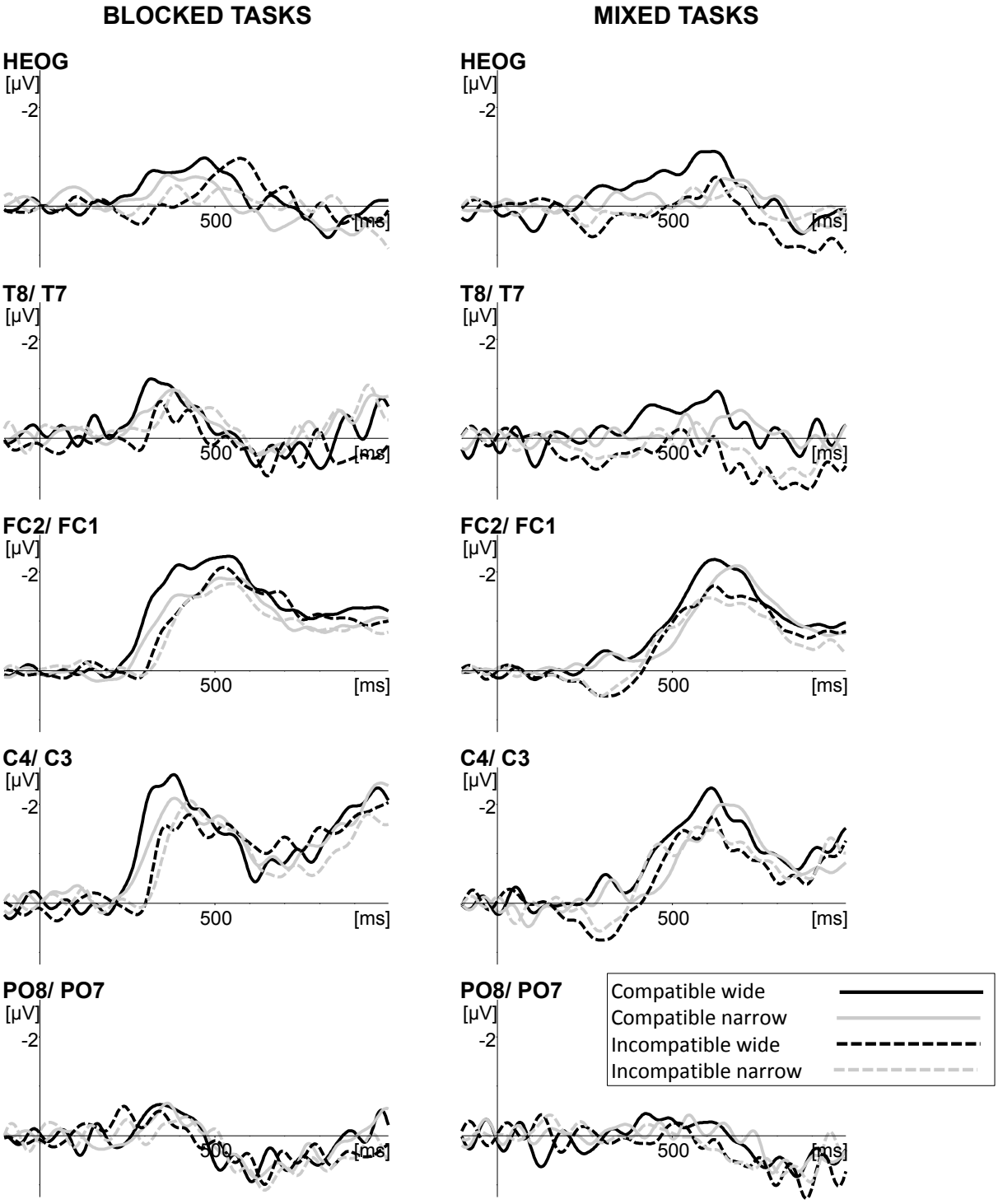

Figure 4.3. Stimulus-locked Lateralized Event Related Potentials (L-ERPs) in blocked tasks (left) and in mixed tasks (right), computed to assess asymmetries in eye movements (HEOG), temporal, fronto-central, central and parietal areas. Signals are depicted separately according to Eccentricity and S-R Mapping.

Prior to LRP onset, preferential response activation was expected in the blocked incompatible task and in mixed tasks, within an interval that commenced later than LRP onset in the blocked compatible condition (234 ms), but earlier than the LRP onset in the relevant condition (within an interval of 200 ms or 51 time-samples). Due to its brief

occurrence (unlike final LRPs), significance of the sequence length of preferential response activation (see Figure 4.5) was assessed by successive t-tests for testing difference waves (Guthrie and Buchwald, 1991). Assuming a signal length of approximately 50 samples and a high autocorrelation (ø = 0.9), a sequence of preferential response activation (significantly different from zero) can be considered significant to a 95% confidence interval if its length is at least 9 consecutive samples. In the blocked incompatible task there was a small (but non-significant) activation of the incorrect response at around 277 ms. In mixed compatible trials, early correct response activation was significant for just 8 time-samples from 313 ms until 340 ms, which should therefore be interpreted as a trend. In mixed incompatible trials, there was a large Gratton dip (incorrect response activation), significant from 258 ms until 352 ms. When accounting for sequential effects in mixed tasks, trials that were preceded by compatible trials led to correct response activation for compatible trials (significant from 297 ms until 359 ms) that subsequently disappeared, and incorrect response activation for incompatible trials (significant from 199 ms until 387 ms). When preceded by incompatible trials, there was no initial correct response activation for compatible trials, but some activation of the incorrect response was visible in a later interval (around 379 ms, ns). When incompatible trials were preceded by incompatible trials, a Gratton dip was significant from 273 ms until 313 ms.

In order to precisely detect the temporal interval of S-R mapping effects in LRPs, difference waves were calculated between LRPs for compatible and incompatible trials, and subsequently subjected to permutation tests (Blair & Karniski, 1993) that produced exact t-values and p-values associated with every time-sample within the segment (from 100 before until 800 ms after the stimulus). In blocked tasks, the interval of the mapping effect in LRPs was between 263 ms and 357 ms. In mixed tasks, the mapping effect was only briefly significant for 3 of the time-samples between 293 ms and 305 ms. When accounting for sequential effects in mixed tasks, following compatible trials the mapping effect was significant in LRPs between 309 ms and 332 ms, but following incompatible trials there was no mapping effect in LRPs.

In sum, final LRPs were delayed in the blocked incompatible task and in mixed tasks, compared to the blocked compatible task. In an interval that commenced later than LRP onset in the blocked compatible task and ended prior to LRP onset in the mixed tasks, early preferential response activation was present in mixed tasks. This preferential response

activation reflected the S-R Mapping in both the current trial (correct response activation for the compatible and incorrect response activation for the incompatible mapping) and preceding trial (following incompatible trials the interval of preferential response activation was shorter or absent). Interestingly, this preferential response activation disappeared before the onset of the final LRP, even in compatible trials preceded by compatible trials.

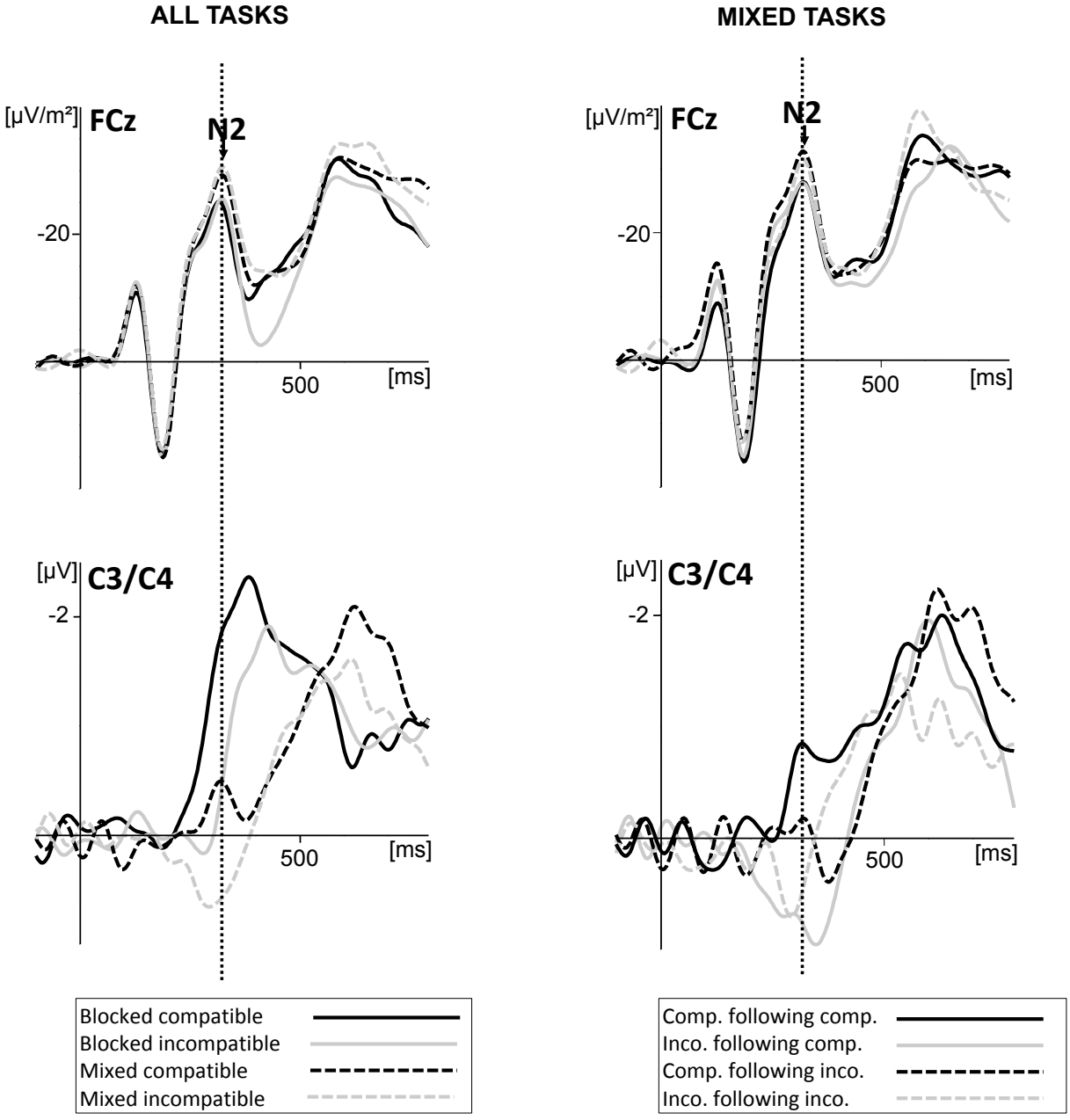

Figure 4.4. Stimulus-locked CSD-transformed ERPs at FCz (top), and stimulus-locked LRPs at C3/C4 (bottom). The left panel depicts grand averaged waveforms in both tasks, separated for S-R Mapping. The right panel depicts grand averaged waveforms in mixed tasks, separated for Previous S-R Mapping and Current S-R Mapping (comp. = compatible S-R Mapping, inco. = incompatible S-R Mapping). The dotted vertical lines highlight the timing of N2 in relation to the LRPs.

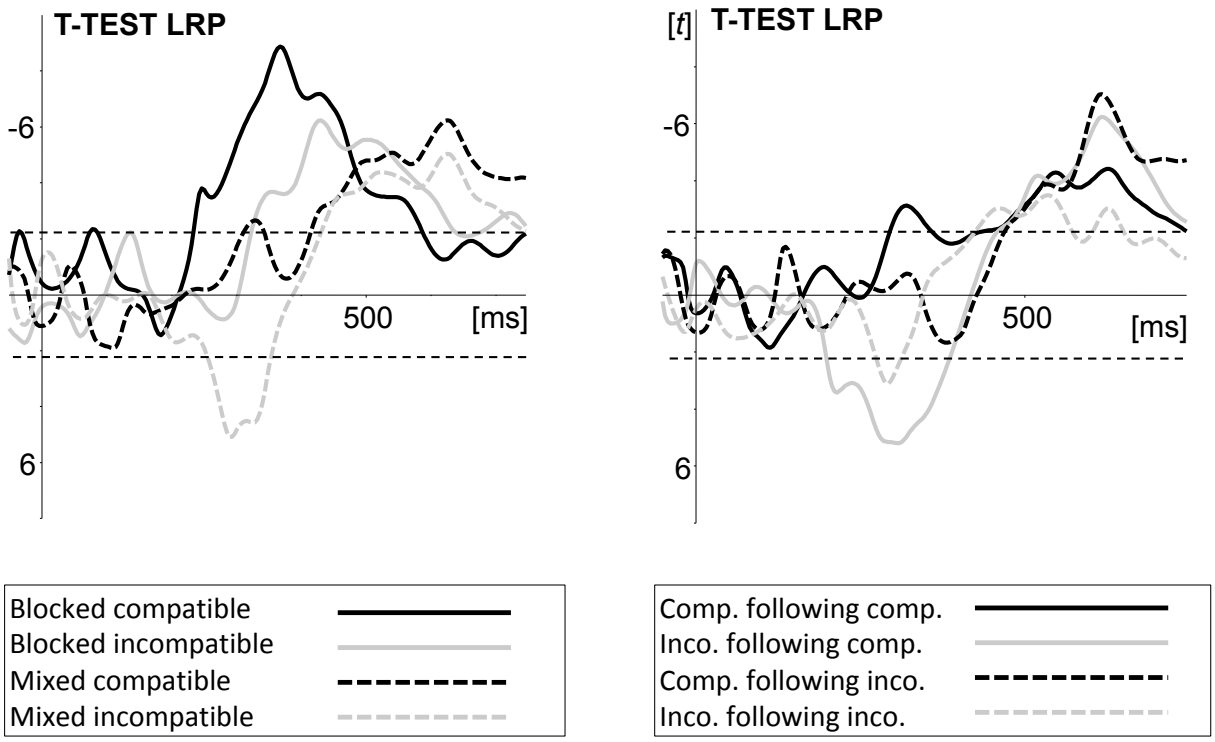

Figure 4.5. Paired t-tests of stimulus-locked LRPs at C3/C4. The left panel depicts t-values in blocked and mixed tasks, separated for S-R Mapping. The right panel depicts t-values in mixed tasks, separated for Previous S-R Mapping and Current S-R Mapping. Dashed horizontal lines depict the 95 % confidence interval ($t(13) = 2.16$, $p = .05$).

4.2.3 Stimulus-locked N2

N2 was maximal at FCz, with a mean latency of 315 ms. No latency effects approached significance. Mean amplitudes per condition are listed in Tables 4.2 and 4.3, and effects are illustrated in Figure 4.4. Mixed tasks (mean -37 $\mu V/m^2$) elicited a larger N2 than blocked tasks (mean -32 $\mu V/m^2$), $F(1,13) = 9.2$, $p = .010$, $\eta_p^2 = .415$. The peak amplitude of N2 in mixed tasks was somewhat larger following incompatible trials, but this difference failed to reach significance ($F = .37$, $p = .56$), as did the interaction between Current and Previous S-R Mapping ($F = 2.3$ $p = .15$).

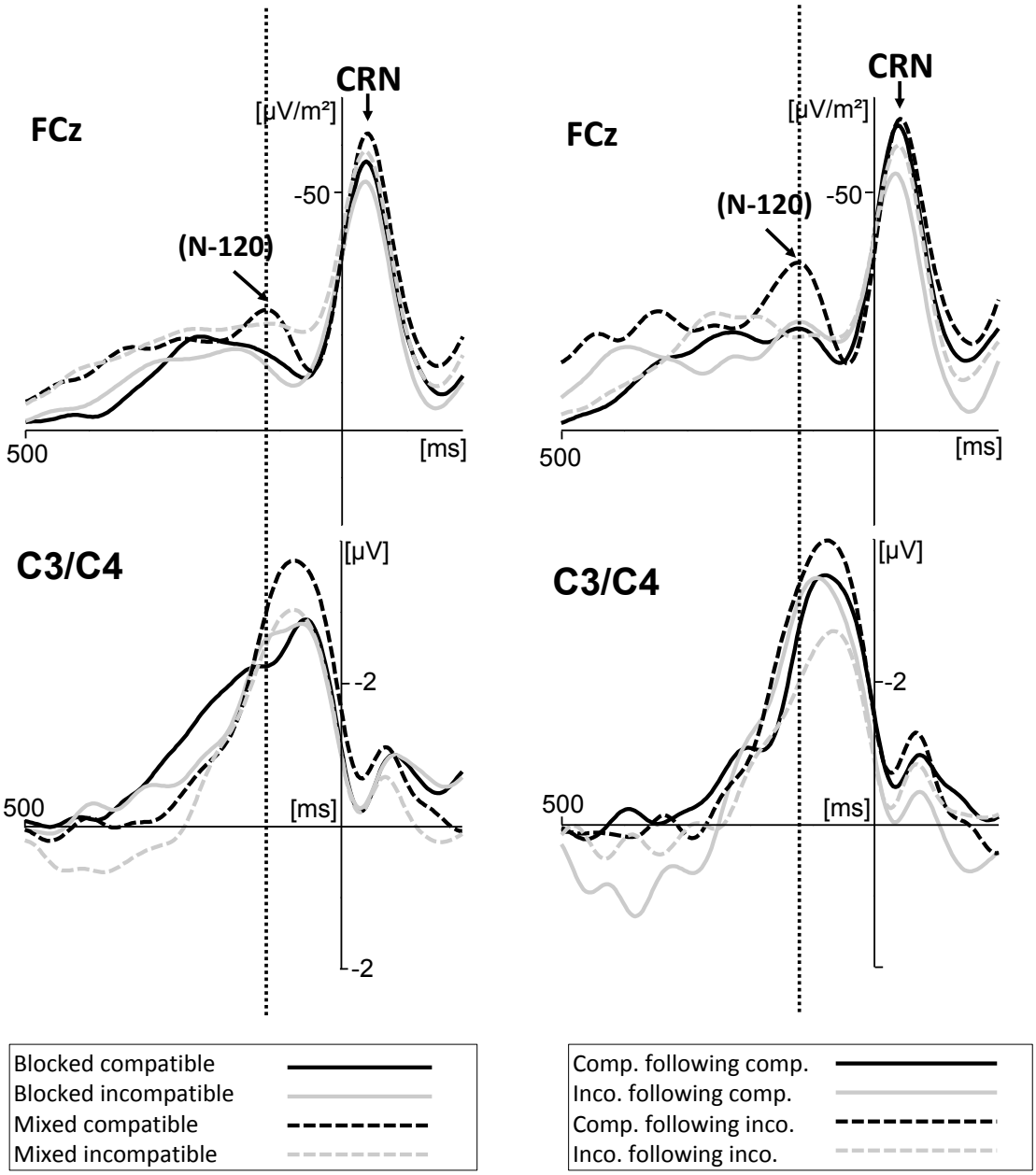

Figure 4.6. Response-locked CSD -transformed ERPs at FCz (top), and response-locked LRPs at C3/C4 (bottom). The left panel depicts grand averaged waveforms in both blocked and mixed tasks, separated for S-R Mapping. The right panel depicts grand averaged waveforms in mixed tasks only, separated for Previous S-R Mapping and Current S-R Mapping. The dotted vertical lines highlight the peak of N-120 in relation to the LRPs.

4.2.4 Response-locked ERPs

Response-locked ERPs (Figure 4.6, upper panel) exhibited an additional component, peaking in grand averaged response-locked ERPs approximately 120 ms prior to the response (from here on N-120). An interaction (trend) between Task and S-R Mapping suggested that N-120 was enhanced specifically in mixed compatible trials ($F(1,13) = 4.5$ $p = .055$, $\eta_p^2 = .255$). Also due to the possibility of temporal overlap between N-120 and N2 in blocked tasks (with

shorter RTs), separate analyses were performed on mixed tasks. N-120 was significantly larger for mixed compatible trials (22 μV/m^2 peak-to-peak) than for mixed incompatible trials (14 μV/m^2 peak-to-peak), $F(1,13) = 5.3$, $p = .038$, $\eta_p^2 = .291$. When accounting for sequential effects, an interaction between Previous S-R Mapping and Current S-R Mapping ($F(1,13) = 11.0$, $p = .006$, $\eta_p^2 = .458$) revealed that N-120 was largest for compatible trials that were preceded by incompatible trials. This interaction was supported by simple effects analyses, in which no S-R mapping effect was present for trials preceded by the compatible mapping (ns), and a reversed S-R mapping effect was present for trials preceded by the incompatible mapping ($F(1,13) = 12.9$, $p = .003$, $\eta_p^2 = .499$). In mixed tasks CRN was enhanced for the compatible mapping (mean 58 μV/m^2 peak-to-peak) compared to the incompatible mapping (mean 47 μV/m^2 peak-to-peak), $F(1,13) = 5.8$, $p = .032$, $\eta_p^2 = .307$. The S-R Mapping in the preceding trial did not influence CRN.

4.2.5 Response-locked LRPs

Response-locked LRPs (Figure 4.6, lower panel) were calculated specifically for mixed tasks to investigate the possibility that N-120 reflected a late enhancement of correct response activation in mixed compatible trials on mapping alternations. An area was selected around the peak of the LRP (between 160 and 40 ms prior to the response), and the mean amplitude in this interval was analyzed by repeated measures ANOVA. An interaction between Previous S-R Mapping and Current S-R Mapping ($F(1,13) = 5.1$, $p = .042$, $\eta_p^2 = .282$) revealed the peak of the response-locked LRP to be largest in compatible trials preceded by incompatible trials, demonstrating the same pattern of results as N-120.

4.2.6 Raster-like Plot

A raster-like plot (Figure 4.7) of the CSD-transformed amplitudes at FCz for participant 6 in mixed compatible trials with the narrow eccentricity revealed two negative components between stimulus and response. N2 appeared from around 300 ms following the stimulus, and N-120 appeared time-locked to and preceding the response.

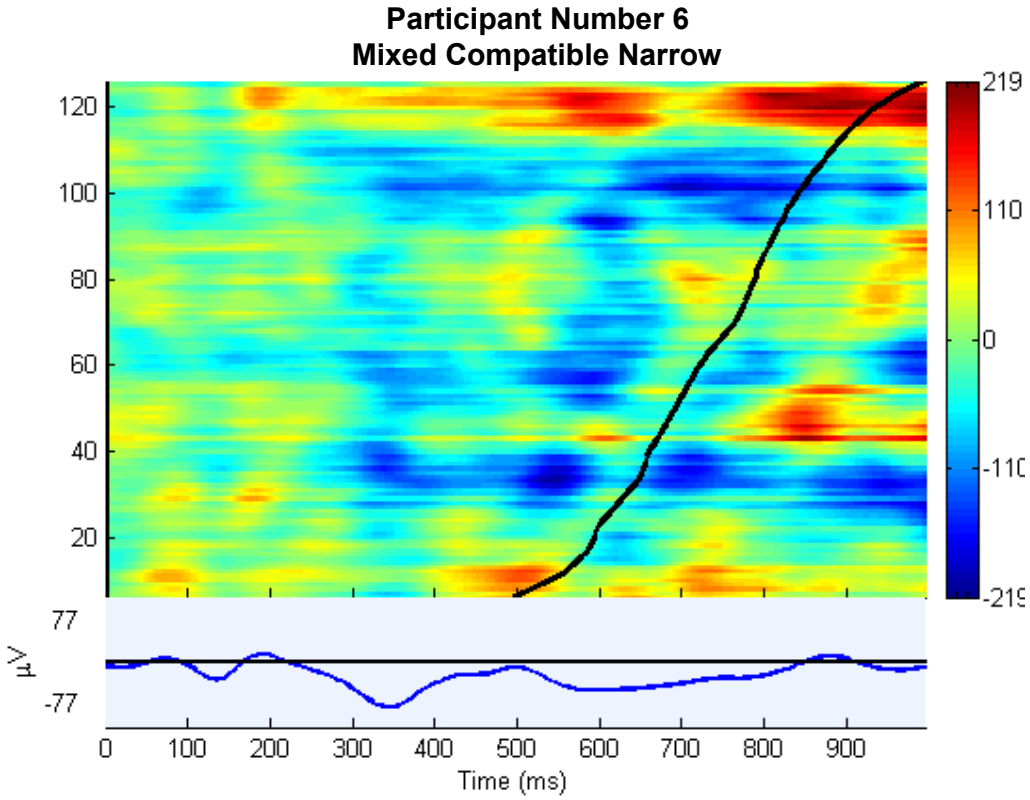

Figure 4.7. Raster-like plot of stimulus-locked CSD-transformed amplitudes at FCz for a representative participant (nr. 6) in the mixed compatible condition with the narrow eccentricity. All individual trials are shown on the vertical axis as a function of increasing RT. Negative amplitudes are depicted in blue and positive amplitudes in red. The vertical axis depicts stimulus onset, and the diagonal black line depicts RT. Stimulus-locked N2 is visible as a negative component from about 300 ms following the stimulus. Response-locked N-120 is visible as an additional negative component that precedes the RT line.

4.3 DISCUSSION

As expected, S-R mapping effects were eliminated in mixed compared to blocked tasks, confirming the findings of previous studies comparing blocked and mixed tasks (Proctor & Vu, 2002; De Jong, 1995; Shaffer, 1965; Stoffels, 1996a; Stoffels 1996b; van Duren & Sanders, 1988; Vu & Proctor, 2004). The compatible-trials-dependent elimination/reversal of SRC effects suggests that performance in mixed S-R mapping tasks is biased towards the incompatible mapping (Jennings et al., 2002). This bias implies a locus in the indirect route of a dual-route model, reflecting logical recoding or task schema competition. In mixed tasks, increased *proactive* cognitive control (reflected at N2) was enhanced in all mixed trials, and is likely involved in the activation of two competing S-R mappings in the indirect route, favoring the incompatible mapping. The bias towards the incompatible mapping is further supported by the presence of a response-locked *reactive* control component (N-120) that is enhanced in mixed compatible trials compared to mixed incompatible trials.

Taking account of the S-R mapping in the preceding trial, we confirmed the prediction that in mixed tasks S-R Mapping effects are present following compatible trials but reversed following incompatible trials. The complete reversal of S-R Mapping effects following incompatible trials provides further support to the assumption that the elimination of S-R mapping costs in mixed tasks can be accounted for by performance differences in the indirect route of a dual-route model. In other words, the spatial response is recoded (e.g., so that left = right) according to the concept of 'logical recoding' (De Jong et al., 1994; Hedge and Marsh, 1975), and this recoding must be overcome when alternating between a compatible and an incompatible mapping. Interestingly, N-120 proved to be most sensitive to compatible trials that were preceded by incompatible trials, implying that late *reactive* control was involved in overcoming activation of the incompatible mapping. Response-locked LRPs supported this interpretation, exhibiting an enhancement of the LRP peak on exactly those trials in which N-120 was also enhanced. Suppression of S-R priming via the direct-route predicted an elimination, but not reversal, of S-R mapping effects. *Proactive* cognitive control at N2 is therefore most likely concerned with initial activation of an S-R mapping, and *reactive* control at N-120 is involved in late correction of the S-R mapping when this has been incorrectly applied.

Even after accounting for trials that were preceded by an incompatible trial, compatible trials were still at a general disadvantage in mixed tasks, suggesting that larger costs are incurred when overcoming such recoding than when initiating it. This specific disadvantage to compatible trials compliments a task schema explanation of mixed task performance (Jennings et al., 2002; Norman & Shallice, 1986), whereby switching to a dominant task incurs larger costs than switching to a less dominant task (Allport, Styles, & Hsieh, 1994). Task-preparatory activity has been investigated with ERPs by Karayanidis, Provost, Brown, Paton, and Heathcote (2011), who found that switching between tasks enhanced a centro-parietal positivity 300-400 ms following the cue. In their study the target was presented later than the cue, but it is difficult in the current study to distinguish which stimulus-locked component might be related to task-switching.

Analyses of LRPs investigated direct S-R priming and indirect conditional response activation. Although the Gratton dip in the blocked incompatible task was small, LRP onset was 90 ms later in incompatible compared to compatible trials. This difference possibly reflects a combination of effects via the direct and the indirect route of a dual-route model, whereby compatible trials benefited from both S-R priming via the direct route, and faster stimulus-response translation via the indirect route. In mixed tasks, LRP onset in incompatible trials was just 20 ms later than in compatible trials, whereby both compatible and incompatible trials exhibited early preferential response activation towards the stimulus-side, which was subsequently eliminated prior to final LRP onset. Considering that early preferential response activation in mixed tasks peaked around the same time as the final LRP onset in blocked incompatible trials, it seems likely that such preferential response activation reflects conditional processing via the indirect route rather than direct S-R priming.

Smaller S-R mapping effects in LRPs in mixed tasks largely reflected a reduction of preferential response activation following incompatible trials. A reduction of S-R priming following incompatible trials in SRC tasks has frequently been interpreted as selective suppression of the direct route (De Jong, 1995; Praamstra et al, 1999; Ridderinkhof, 2002; Stoffels, 1996b; Stürmer et al., 2002, 2007). However, considering that preferential response activation in mixed tasks occurred around the same time as LRP onset in the blocked incompatible task, we interpret the reduction in preferential response activation as mainly indirect-route effects. We cannot rule out the possibility of suppression of S-R priming in

mixed tasks, but it cannot account for the presence of incorrect response activation in mixed incompatible trials preceded by incompatible trials. It is sufficient to say that indirect-route effects are essential to an account of performance in mixed tasks, and that suppression of S-R priming plays no more than a minor role in such an account.

Most interesting was the pattern in LRPs for mixed compatible trials, in which an early activation of the compatible response abruptly disappeared before the onset of final response activation. This elimination was even present when the preceding trial was also compatible. The disappearance of the early correct response activation might be interpreted as a *late voluntary* suppression of the direct route. However, this seems unlikely, due to the same late reduction of correct response activation following compatible trials. Alternatively, this might be interpreted as an *involuntary* automatic inhibition of an activated response, similar to the findings of Eimer and Schlaghecken (1998), exhibiting unconscious inhibition following subliminal primes. But this interpretation also seems unlikely due to the later findings of the same authors (Eimer & Schalghecken, 2002) that such automatic inhibition was not present for consciously perceived stimuli. A third possibility is that early automatic S-R priming simply decays prior to the onset of final controlled response activation, similar to the response activation observed for spatial cues that do not require a response (e.g. Eimer, 1995; Eimer, Hommel, & Prinz, 1995). However, in the current task the spatial stimuli always require a response, and activation via the indirect route is already influential during this interval. Having cast doubt on three alternative interpretations of the brief disappearance of S-R priming in mixed compatible trials, it seems most plausible that the pattern in LRPs reflects conditional processing via the indirect route. The next issue that needs to be resolved concerns the extent to which such conditional processing reflects cognitive control.

As predicted, fronto-central N2 exhibited larger amplitudes for mixed tasks than for blocked tasks, suggesting increased *proactive* cognitive control. The response conflict hypothesis predicts that there will be increased control following detection of conflict (Carter & van Veen, 2007; Botvinick, Braver, Carter, Barch, & Cohen, 2001). Considering the difficulty of assessing which trials involved the most conflict in mixed tasks, it is only safe to assume that increased interference was involved on S-R mapping alternations. When accounting for the S-R Mapping in the preceding trial, grand averaged ERPs suggested an enhancement following incompatible trials, and when alternating between S-R Mappings, but neither of these effects verged on significance. Control at N2 in mixed tasks might

involve suppression of an already active task schema (c.f. Jennings et al, 2002), or perhaps a general suppression of all response activation accumulated via both direct and indirect routes (c.f., Band & van Boxtel, 1999; Jennings & van der Molen, 2005). But overall, proactive control reflected at N2 is most likely related to the activation of an S-R mapping, possibly with the involvement of suppression of task schemas or accumulated response activation.

Interestingly, N-120 was particularly enhanced when alternating from an incompatible to a compatible trial, which were the trials with the slowest RTs, the most errors, and the latest LRP onset. Importantly, a raster-like plot of all trials in the mixed compatible condition with the narrow eccentricity for one representative participant clearly defined N-120 as a unique component that varied with and preceded the response. The slow RTs and enhanced response-locked LRP peak in compatible trials preceded by incompatible trials support the interpretation of N-120 as an additional control mechanism that activates the correct S-R mapping just prior to the response. We therefore assume a *proactive* bias towards the incompatible mapping (Jennings et al., 2002), whereby N-120 reflects *reactive* control that later reinforces the appropriate S-R mapping. This finding implies dissociation between N-120 and CRN. CRN was sensitive to mixed compatible trials, but not to S-R mapping alternations. Assuming that both ERN and CRN reflect the degree of incorrect response activation, it makes sense that CRN is enhanced in trials that frequently result in full errors. However, the fact that CRN was not sensitive to the S-R Mapping in the preceding trial suggests that much of the interference expected when alternating between S-R mappings was resolved by N-120 prior to the response. An alternative account is offered by Bartholow and colleagues (2005), who interpreted an enhanced CRN as detection of an inappropriate strategy. Assuming a strategic bias towards the incompatible mapping, the latter account is supported by the sensitivity of CRN to mixed compatible trials, but not by its insensitivity to S-R mapping alternations, in which an inappropriate strategy is more likely.

The response conflict hypothesis relies upon simulation for assessing the amount of conflict that leads to cognitive control (Carter & van Veen, 2007; Botvinick, et al., 2001; Yeung et al, 2004). However, an account of the control mechanisms involved in resolving interference in mixed S-R mapping tasks does not need to depend upon assessing the amount of conflict, particularly considering the various stages in which conflict might be produced via both the direct and the indirect route. Rather, the increased control in mixed

tasks is apparent in the combined results from behavioral and electrophysiological measures. Overall, the pattern in LRPs suggests that the S-R mapping from the preceding trial is still partially active in the indirect route. The enhanced N2 in mixed tasks supports the assumption that *proactive* control (as manifested by N2) initially selects the S-R mapping. However, when two S-R mappings are competing, *proactive* control is not always accurate in its selection. The performance and ERP results all highlight a particular disadvantage to compatible trials preceded by incompatible trials, supporting the assumption of a proactive bias towards the incompatible mapping in mixed S-R mapping tasks. However, a *reactive* control mechanism (as manifested by N-120), particularly enhanced on an alternation of S-R mappings, seems capable of correcting erroneous response activation immediately prior to the response. In sum, *proactive* control activates the S-R mapping via the indirect route of a dual-route model, and *reactive* control reinforces the appropriate S-R mapping when needed.

We can differentiate three ERP components related to cognitive control, by their timing and by their unique pattern of effects. In investigating the mechanisms of *proactive* control, we have found that N2 is equally enhanced in all mixed trials, relatively independent of the S-R mapping in the current or the preceding trial. In terms of *reactive* control, we have confirmed that such control is especially effective when alternating between S-R mappings. This finding is highly relevant to task-switching studies, and further research with mixed SRC tasks might reveal the underlying mechanisms of *reactive* control. Finally, future simulation studies might reveal which trials involve the most conflict in mixed S-R mapping tasks, and whether either *proactive* or *reactive* cognitive control mechanisms are influenced by the amount of conflict in the current or preceding trial.

Chapter 5:

Proactive and reactive control over lateralized motor competition: A Laplacian ERP analysis

The current study investigated the dynamic interplay between proactive and reactive control using response-locked ERPs. We manipulated the probability of compatible vs. incompatible stimulus-response (SR) mappings to test two hypotheses. Firstly we anticipated that participants prepare (proactively) for the expected SR-mapping, predicting that on unexpected trials reactive control corrects the mapping, appointing inhibition of the incorrect response (indexed by ipsilateral positivity) and activation of the correct response (indexed by contralateral negativity). Secondly we anticipated that when expecting an incompatible SR-mapping, response activation would initially be decreased, leading to a late enhancement to contralateral negativity. Behavioral and electrophysiological measures confirmed that reactive control was most prominent on unexpected compatible trials. However, ipsilateral positivity was most enhanced in unexpected incompatible trials. Contralateral amplitudes suggested an initial bilateral inhibition in unexpected trials. The mechanisms of reactive control seem to depend upon strategic performance adjustments by proactive control.

2.5 INTRODUCTION

A recent model that differentiates between dual-mechanisms of cognitive control (Braver, Gray, & Burgess, 2007; De Pisapia & Braver, 2006) assumes that in order to account for variation in task performance, proactive and reactive cognitive control should be distinguished from one another. According to the Dual Mechanisms of Control (DMC) model, proactive control involves preparatory attention and depends on global cues that are available prior to stimulus presentation, such as preparatory cues or task settings. Reactive control involves late correction/resolution of competition between active responses, and depends on information that cannot be predicted in advance. The amount of proactive and reactive control applied is assumed to depend upon both the individual and the environment, which might alter the balance between a proactive vs. a reactive cognitive control strategy (Braver, 2012). The current study uses behavioral and brain potential measures to investigate the interplay between proactive and reactive cognitive control, and specifically the mechanisms underlying reactive control.

2.5.1 Stimulus Probability

One way of inducing proactive (preparatory) control is by manipulating the probability of a stimulus or response between blocks, so that participants are likely to prepare for the expected event. This comparison was made by Gratton et al. (1992, Experiment 2), who asked participants to perform an Eriksen flanker task (Eriksen & Eriksen, 1974), in which a central target letter was flanked by noise stimuli (also letters) that were either congruent or incongruent to the response. Experimental blocks were biased towards congruent arrays (75%), towards incongruent arrays (75%), or unbiased (50%). A similar task was used by Bartholow et al. (2005) with 80%/20% probabilities in the biased conditions. In both studies responses were faster and more accurate to congruent vs. incongruent arrays, but this effect of noise was largest in blocks with a congruent bias and smallest in blocks with an incongruent bias. One hypothesis that might account for the greater noise effect when expecting a congruent array is the possibility that participants can influence the level of response activation in preparation for the stimulus, bringing it closer to a threshold that triggers the response (Niemi & Näätänen, 1981; see also Brown & Heathcote, 2005; Jahfari et al., 2012; Hanes & Schall, 1996). It is likely that preparatory (proactive) control enabled

faster responding when expecting congruency, at the expense of slower responding and increased errors for unexpected incongruent arrays.

Similarly, a higher likelihood of an incongruent array may result in proactive control reducing response activation. This hypothesis was supported in an experiment by, Klein et al. (2014), who used an Eriksen task during which motor-evoked potentials (MEPs) were elicited using transcranial magnetic stimulation (TMS), and found that in blocks in which 80% of the arrays had incongruent noise, MEPs were already suppressed at stimulus onset. Assuming that proactive control is able to strategically increase or decrease the level of response activation depending on the expected event, reactive control is needed to compensate for proactive control measures when expectancies are violated. In experimental settings, reactive cognitive control is assumed to act upon information that is only available after presentation of the target stimulus. The conflict-monitoring theory proposes that cognitive control in general, which is carried out via the prefrontal cortex (PFC), depends upon conflict monitoring via anterior cingulate cortex (ACC) (Botvinick et al, 2001). Within this context, Yeung et al. (2004) showed that conflict evaluation via ACC is reflected in brain potential measures by two electrophysiological components. The first component, N2, is a negative component that precedes the response and is related to cognitive control (for a review, see Folstein & van Petten, 2008). The second component, called Error Negativity (Ne , Falkenstein et al., 1991) or Error Related Negativity (ERN, Gehring et al., 1993) is an increased negativity found following the response in error trials. Yeung et al. assumed that the amount of conflict detected by ACC reflected the amount of competition between active responses. Collectively, these findings support the hypothesis that reactive control involved in response selection depends upon evaluation of competition between active responses.

2.5.2 Task Probability

As a means to examining the interplay between proactive (preparatory) and reactive control, the probability of an SR-Mapping (task-bindings) can be manipulated between experimental blocks. When SR-Mappings are mixed in a spatial task, there are two stimulus dimensions that are relevant to the decision; one dimension (e.g. color) cues the mapping (compatible/incompatible), and the other dimension (e.g. arrow direction) cues the response side, which depends also on the mapping. By manipulating the probability of the SR-Mapping, the predictability of SR-Mapping can be increased, but the response-hand is

still unpredictable (assuming 50% left/right). Therefore proactive control can be assessed by comparing responses associated with predictable vs. unpredictable SR-Mappings, and reactive control can be assessed by comparing responses to unexpected vs. expected stimuli.

Previously, Lungu et al. (2007) used behavioral and brain imaging measures to investigate the effects of SR-Mapping probability on executive control processes carried out via PFC. In unbiased blocks (50/50), reaction times (RTs) and errors were similar for compatible and incompatible mappings, whereas activation of right lateral PFC and anterior ACC were greater for the incompatible mapping. In blocks with either a compatible or incompatible bias (80/20), the most probable mapping in the block was related to better performance and greater activation in medial PFC, whereas the least probable mapping in the block was related to worse performance and increased activation in caudal ACC. The findings in the unbiased blocks support the conflict-monitoring hypothesis of cognitive control (Botvinick et al., 2001), because PFC and ACC are most active on incompatible trials. However, in the probability blocks, activation is enhanced in PFC for the expected SR-Mapping trials and in ACC for the unexpected trials. This pattern of results suggests that in the biased blocks participants (proactively) maintained activation of the expected mapping (via medial PFC), and that reactive control adjusted proactive control when conflict between SR-Mappings was detected (via caudal ACC).

2.5.3 Mechanisms of Control

Assuming that reactive control aims at producing a correct response under conditions of high-conflict, such control might incorporate either late *activation* of the *correct* response and/or late *inhibition* of the *incorrect* response. Recent research with choice RT tasks has investigated the roles of activation and inhibition of primary sensory motor cortex (SM1), using current source density (Laplacian transformed) estimates of scalp potentials (Meckler et al., 2010; Praamstra & Seiss, 2005; van de Laar et al., 2012; Vidal et al., 2003). Just prior to contraction of the muscle that executes the response, a negative-going wave is evident contralateral to the response hand, briefly preceded by a positive-going wave ipsilateral to the response hand. There is evidence from intracranial recordings of the human motor cortex that the contralateral negativity represents activation of the response (Ikeda et al., 1995; Neshige et al., 1988). Interestingly, ipsilateral positivity is only apparent in tasks involving a choice between a left or right hand response, as opposed to simple RT tasks

involving just one hand (Burle et al, 2004; Carbonnell et al., 2004). Therefore Burle et al. reasoned that ipsilateral positivity represents a strategic process involved in inhibition of an erroneous response. Inhibition of an active incorrect response is a process that could be triggered by reactive cognitive control.

The hypothesis that ipsilateral positivity is associated with the inhibition of errors was tested by Meckler et al. (2010) by using a choice RT task, in which the probability of left- versus right-hand responses was manipulated between blocks. In this task, participants are likely to prepare for the expected response hand. The authors found that unexpected responses lead to increased ipsilateral positivity, and that this positivity was largest for the most accurate participants. The authors assumed that ipsilateral positivity represented successful inhibition of the prepared incorrect response. In terms of proactive and reactive control, the results of their study are consistent with the notion that proactive control raises the level of response activation in preparation for the expected response (c.f. Niemi & Näätänen, 1981), while reactive control triggers inhibition of the expected response when an unexpected response is required.

Meckler et al. (2010) also reported a negative frontocentral component (N-40) just prior to the onset of muscle contraction in unexpected and unbiased (50% probability) trials. The authors suspected that the process underlying N-40 was the agent implicated in the inhibition of the expected response manifested by ipsilateral positivity. Unfortunately, N-40 could not be scored for expected trials, precluding a test of its sensitivity to the probability manipulation. Considering the simplicity of the task, participants were able to fully prepare the expected response; thus, the agent implicated in response inhibition was possibly redundant in expected trials. It is likely, then, that N-40 was absent on expected trials because response selection is not needed on those trials.

Another candidate for the electrophysiological signature of reactive control is response-locked N-120 (Mansfield et al., 2012). Employing a spatial mapping task, SR-Mapping was either blocked (predictable, one mapping per block) or mixed (unpredictable, both mappings in the same block). For behavioral measures, the SR-Mapping effect observed for predictable blocks was eliminated in mixed blocks. We found global effects (between blocks) on the amplitude of stimulus-locked N2, which we assumed to reflect activation of an SR-Mapping in the mixed task. We also identified a response-locked frontocentral midline

negativity (N-120), which peaked 120 ms prior to the correct response and was especially enhanced for compatible trials following incompatible trials. It appeared that in mixed blocks participants were inclined to prepare (proactively) the incompatible mapping, leaving compatible trials at a disadvantage and in need of reactive control adjustments. Response-locked N-120 was both functionally and temporally independent from stimulus-locked N2, and therefore interpreted to reflect reactive control involved in correction of the SR-Mapping. N-120 bears similarities to the N-40 reported by Meckler et al. (2010). N-40 was time-locked to electromyographic (EMG) onset, and Meckler et al. report a delay of 80-90 ms between EMG onset and response onset. In other words, if time-locked to the response this component would be an N-120. It seems likely that EMG-locked N-40 and response-locked N-120 reflect the same underlying mechanism of reactive cognitive control.

2.5.4 Hypotheses

The current study aims to investigate the appointment of contralateral activation and ipsilateral inhibition by reactive control, under varying degrees of proactive control. According to the DMC (Braver, 2012), a proactive control strategy is favored with high expectancy, and a reactive control strategy is favored with low expectancy. Therefore, we assume that a reactive control strategy will dominate in unbiased blocks, and a proactive strategy will dominate in biased blocks. Also, proactive control is assumed to increase with the expectation of conflict (Braver, 2012). Therefore, in biased blocks we expect more proactive control with an incompatible bias compared to a compatible bias. If proactive control is enhanced in blocks with an incompatible bias, then unexpected compatible trials should demonstrate the worst performance and compatible (not incompatible) trials should suffer the greatest sensitivity to the direction of the bias. We assume that reactive control will depend upon the suitability of the proactive control applied to the task. Because in this task the response hand is never predictable, reactive control should be involved in all trials, but minimal when the correct mapping is prepared by proactive control (in expected trials), and most enhanced when the wrong mapping is prepared (in unexpected trials). We expect performance deficits on unbiased compared to expected trials, and in unexpected compared to unbiased trials. Activating the unexpected mapping and response hand will take longer if response activation has been reduced. This will be manifested in a late enhancement to contralateral negativity on unexpected compatible trials. In brief, we hypothesize that

unexpected compatible trials will exhibit the largest performance deficits and most enhanced reactive cognitive control (reflected in the amplitude of ipsilateral positivity, contralateral negativity, and response-locked N-120).

2.6 METHODS

2.6.1 Participants

Behavioral and electrophysiological data were collected from seventeen psychology students, in exchange for course credit. Three participants were excluded from analyses due to excess artifact in the EEG. The remaining fourteen participants (13 women and 1 man) were all right-handed, with ages ranging from 18 to 23 years (mean 19 years). All were neurologically healthy, had normal or corrected-to-normal vision, and gave informed consent for their participation in the experiment.

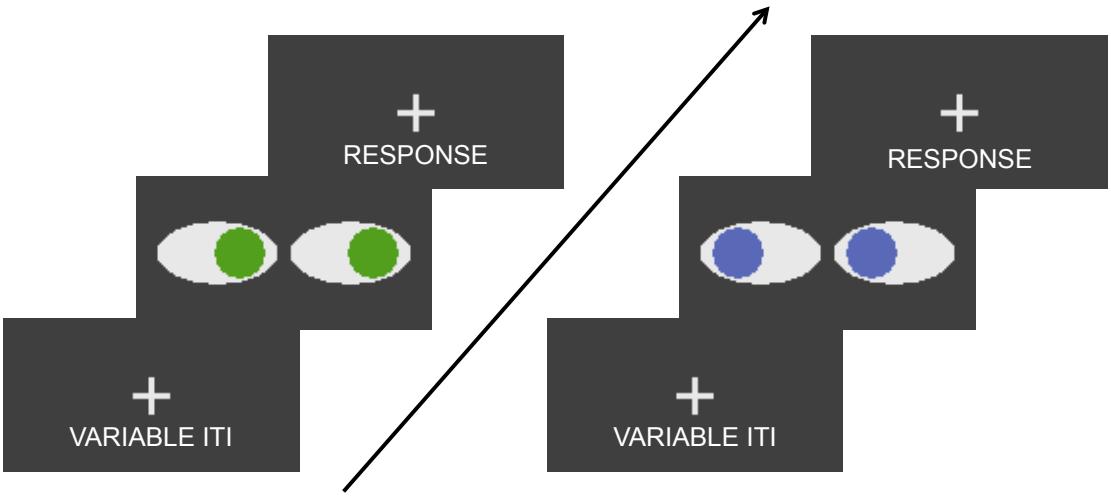

Figure 5.1. Examples of two trial types with a detail of the gaze stimuli used in the experiment**.**

2.6.2 Stimuli and Apparatus

The stimuli (see Figure 5.1) were a schematic pair of eyes, with a height of 1.0 cm and total width of 3.6 cm (2.9 visual degrees). The white eye stimuli (each 1.7 cm wide, 1.4 degrees, with 2 mm space between the eyes) were centered on a dark gray background. The colored pupils (7 mm diameter) were positioned in the left or right half of each eye to form each gaze direction. The pupils were blue or green, each color with the same luminance. Participants were seated in a dimly lit, sound-attenuated, electrically shielded room, and a 14-inch monitor was positioned at eye-level at a distance of 70 centimeters.

2.6.3 Tasks and Procedure

Participants were required to respond as quickly and accurately as possible according to both the color and gaze direction of the stimuli, using their index fingers to press one of two designated response buttons. Gaze direction cued the response hand, but eye color cued the mapping (compatibility), such that on incompatible trials participants were required to respond with the hand opposite to the direction of the eye gaze. Half of the participants gave a compatible response to green eyes and an incompatible response to blue eyes. The other half of the participants received the opposite color-mapping instruction. Three Probability conditions were included in the experiment (80%, 50%, and 20% compatible), the order of which was counterbalanced across participants. The number of left- and right-handed responses was always equal within each block. For each of the three Probability conditions, participants first completed a practice session (40 trials) before completing two consecutive measurement blocks of 200 trials (approximately 10 minutes each). A fixation cross (+) was always present, except for during stimulus presentation. Each new trial began with a variable inter-trial interval, between 1600 ms and 3100 ms (in steps of 1 ms), followed by the presentation of the gaze stimulus for 150 ms.

2.6.4 EEG Recording and Analysis

EEG was recorded from 128 locations using active sintered electrodes (BioSemi Active 2) mounted in an elastic cap, with a sample rate of 512 Hz, plus two mastoid reference electrodes. Electrode locations are reported in line with the five percent electrode system described by Oostenveld and Praamstra (2001). Vertical eye movements were recorded from electrodes positioned above and below the right eye, and horizontal eye movements from electrodes at the outer canthus of each eye. Raw data were referenced off-line to average mastoids and all channels were band-pass filtered from 0.1-30 Hz. The filtered data were initially segmented into epochs ranging from 300 ms prior to the stimulus until 1 second after the stimulus, and coded according to condition and accuracy. Under visual inspection, segments with large artifacts (2% of segments) were removed manually. Blinks (in 69% of segments) were detected and corrected using the regression technique by Gratton and Coles (1983). Horizontal eye movements were calculated as “right – left”, and this difference wave was subjected to the same automatic rejection procedure as the remaining EEG channels, which rejected every segment that had a voltage step larger than 100 μV, or an amplitude

range of more than 120 μV, on any channel (10% of segments). Segments were baseline corrected to the 100 ms prior to stimulus presentation. These epochs were then segmented time-locked to the response, ranging from 500 ms prior until 300 ms after the response. Stimulus- and response-locked ERPs were averaged for each participant per condition, excluding errors and outliers. Each of the conditions (Bias x SR-Mapping) was represented by a maximum of 320, 200, or 80 trials (expected, unbiased, and unexpected trials), and a minimum of 24 trials per participant. In order to calculate the surface Laplacian, for the stimulus- and response-locked ERPs, a CSD transformation (Perrin, Pernier, Bertrand, & Echallier, 1989, 1990), with order of splines 3, and maximum degree of polynomials 15, was performed on the participant's condition means. This procedure transforms the surface EEG based on spherical splines, eliminating the need for an external or average reference (see also Ferree, 2006). Subsequently, an additional low-pass filter (15 Hz) was carried out prior to scoring the ERPs.

Contra- and ipsilateral waveforms (Figure 5.3) were calculated for response-locked ERPs for three symmetrical electrode pairs above the motor cortex: C3/C4, C3'/C4', and C1/C2 (see Homan, Herman, & Purdy, 1987, for the location of brain areas relative to the 10-20 electrode system), by first coding contra/ipsi lateral activity separately for left- and right-hand responses, and then averaging contra/ipsi lateral activity over left- and right-hand responses. Following inspection of grand averaged contra- and ipsi-lateral waveforms, mean ipsilateral amplitudes were exported in two separate intervals prior to the response; the first at the start of the positive-going wave (180–80 ms), and the second at the peak of ipsilateral positivity (75-35 ms). Contralateral amplitudes were scored as the average amplitude between 75 and 35 ms prior to the response. Response-locked N-120 was scored at FFCz as a peak-to-peak measure by first performing a baseline correction to the positive peak at the response, separately for each participant. The N-120 peaks were picked by hand, ranging from 211 until 52 ms pre-response. Peak-to-peak measures were used due to the presence of clear N-120 peaks but considerable variability in timing.

2.6.5 Statistical Analyses

Outliers (trials with an RT more than 2 SDs outside the participants' condition mean) and errors were omitted from analysis of RTs and ERPs. In order to assess the interplay between proactive and reactive control, each of the measures (RTs, error percentages, contralateral

amplitudes, ipsilateral amplitudes, and N-120 peak amplitudes) was analyzed by repeated measures analysis with the factors Bias (compatible bias, no bias, incompatible bias) and SR-Mapping (compatible, incompatible). In order to assess whether proactive control involved preparation of the expected mapping, for all measures paired t-tests were used to make direct comparisons between expected and unbiased trials, and between unbiased and unexpected trials, separately for each mapping. In order to assess the sensitivity of each mapping to Bias/Frequency (expected, unbiased, unexpected), for all measures paired t-tests compared compatible to incompatible trials in each frequency condition. In order to assess whether proactive control was more enhanced with an incompatible bias than with a compatible bias, for all measures, paired t-tests (two-tailed) compared unexpected compatible with unexpected incompatible trials. In accordance with the number of tests performed, interpretations of t-tests were Bonferroni corrected, such that effects were only interpreted as significant when $p<.01$.

2.7 RESULTS

2.7.1 Performance Measures

Performance measures are summarized in the first two columns of Table 5.1. Figure 5.2 illustrates the overall means and standard errors for reaction times (RTs, left) and percentage of errors (right), according to Probability Bias (the percentage of compatible trials in the block) and SR-Mapping. As expected, the SR-Mapping effect was sensitive to Bias on analysis of errors (interaction, $F(2,12) = 10.3$, $p=.003$, $\eta_p^2 = .631$) and RTs (interaction, $F(2,12) = 85.1$, $p<.001$, $\eta_p^2 = .934$). Separate analyses per Bias revealed the standard SR-Mapping effect in blocks with a compatible bias on RTs ($F(1,13) = 110.1$, $p<.001$, $\eta_p^2 = .894$) and Errors ($F(1,13) = 7.3$, $p=.018$, $\eta_p^2 = .359$); no SR-Mapping effect in unbiased blocks on RTs ($F<1$, $p > .6$) and only a trend suggesting a reversed mapping effect on Errors ($F(1,13) = 4.5$, $p=.053$, $\eta_p^2 = .259$); and a significant reversed mapping effect in blocks with an incompatible bias on RTs ($F(1,13) = 51.4$, $p<.001$, $\eta_p^2 = .798$) and Errors ($F(1,13) = 19.2$, $p=.001$, $\eta_p^2 = .596$). Separate analyses per SR-Mapping confirmed the sensitivity of the compatible mapping to Bias, such that reducing the percentage of compatible trials within the block led to slower RTs (Bias, $F(2,12) = 18.5$, $p<.001$, $\eta_p^2 = .755$) and more errors (Bias, $F(2,12) = 8.0$, $p=.006$, $\eta_p^2 = .573$). Analysis of incompatible trials confirmed that reducing the percentage of

compatible trials within the block did lead to less errors on incompatible trials (Bias, $F(2,12) = 4.0$, $p=.047$, $\eta_p^2 = .400$), but the reduction in RTs was only a trend ($F(2,12) = 3.7$, $p=.056$, $\eta_p^2 = .381$).

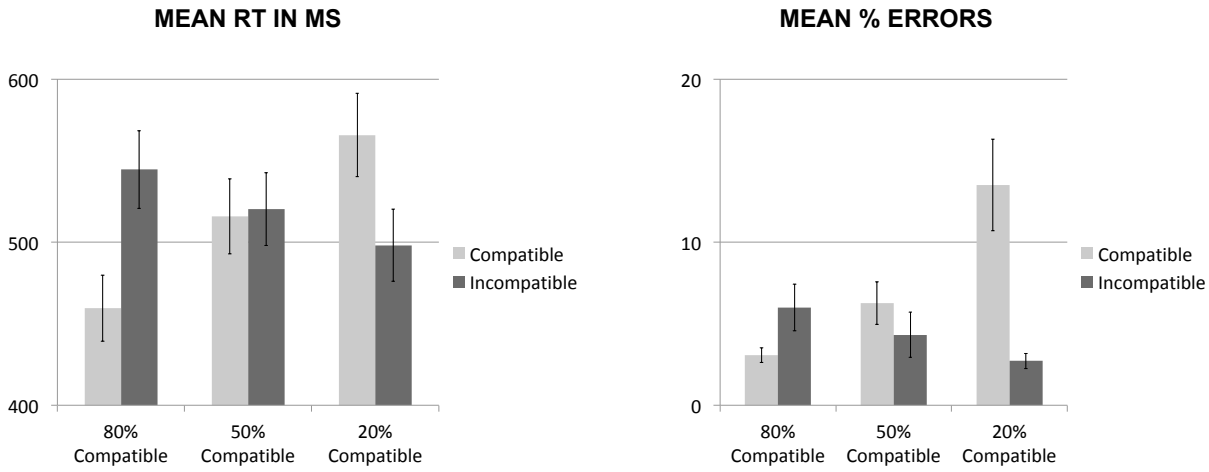

Figure 5.2. Graphical illustration of means and standard errors for RTs in ms (left) and Error percentages (right) in each condition.

We predicted performance effects due to preparation of the expected mapping by proactive control in biased blocks. As expected, unpredictable trials suffered performance deficits compared to expected trials, confirmed by paired t-tests for the compatible mapping on RTs ($t(13) = 4.84$, $p<.001$) and errors ($t(13) = 3.27$, $p = .006$), but not for the incompatible mapping on RTs ($t(13) = 2.56$, $p = .024$) or errors ($p > .1$). Unexpected trials also suffered performance deficits compared to unpredictable trials, for the compatible mapping on RTs ($t(13) = 5.43$, $p<.001$) and errors ($t(13) = 3.31$, $p = .006$), but not for the incompatible mapping on RTs ($t(13) = 2.18$, $p = .048$) or errors ($t(13) = 2.36$, $p = .034$). We predicted that compatible trials would suffer most in unexpected trials, due to enhanced proactive control with an incompatible bias. Although this prediction is supported by the greater sensitivity to Bias for compatible trials, the disadvantage to unexpected compatible compared to unexpected incompatible trials was marginal for errors ($t(13) = 3.04$, $p = .010$), and not significant for RTs ($t(13) < 1$, $p > .1$). In sum, we found support for the prediction that specifically the *compatible* mapping is sensitive to the bias in the block.

Probability Bias	Condition	RT ms	Error %	Ipsilateral 330-290 C3/C4	Ipsilateral 180-80 C3/C4	Ipsilateral 75-35 C3/C4	Contralateral 180-80 C3/C4	Contralateral 75-35 C3/C4	Contralateral 75-35 C1/C2	Midline N-120 Amp. FFCz
Bias Comp.: 80% Compatible	Expected compatible	459	3	-3	10	27	2	12	-4	-16
	Unexpected incompatible	545	6	8	24	40	15	24	-6	-22
No Bias: 50% Compatible	Unbiased compatible	516	6	-2	13	32	8	17	-7	-22
	Unbiased incompatible	520	4	-3	8	25	5	15	-10	-15
Bias Inco.: 20% Compatible	Unexpected compatible	566	14	-5	24	37	11	10	-12	-39
	Expected incompatible	498	3	0	5	20	4	12	-12	-18

Table 5.1. Grand averages for all measures in all conditions.

2.7.2 Electrophysiological Measures

Mean amplitudes per condition for all electrophysiological measures are reported in Table 5.1. Figure 5.3 depicts response-locked waveforms at contralateral (left), ipsilateral (right), and midline (FFCz) electrodes for compatible trials (top panel) and incompatible trials (bottom panel), with separate waveforms according to Bias. Figure 5.4 depicts both contralateral negativity and ipsilateral positivity in the biased conditions.

2.7.2.1 Ipsilateral Amplitudes

As expected, at C3/C4 ipsilateral positivity was visible prior to the response, starting at approximately 250 ms and peaking between 80 and 30 ms pre-response. We initially analyzed ipsilateral positivity in two intervals, one at the beginning of the positive wave (180-80 ms pre-response) and one at the peak of the positive wave (75-35 ms). As expected, ipsilateral positivity was most enhanced in unexpected trials, confirmed by an interaction between Bias and SR-Mapping on analyses of the start of the positive wave ($F(2,12) = 4.1$, $p=.044$, $\eta_p^2 = .407$) and of the peak of ipsilateral positivity ($F(2,12) = 5.5$, $p=.020$, $\eta_p^2 = .479$). We expected the largest reactive control measures for compatible trials when the bias was incompatible. In line with this prediction, separate analyses according to Bias revealed that the enhancement to unexpected compared to expected trials was only significant with an *incompatible* bias, on analysis of the start of the wave ($F(1,13) = 8.5$, $p=.012$, $\eta_p^2 = .395$) and a trend on analysis of the peak of ipsilateral positivity ($F(1,13) = 4.4$, $p=.056$, $\eta_p^2 = .253$). With a *compatible* bias (and no bias), there was no difference between compatible and incompatible trials in ipsilateral positivity in either interval (all ps > .1). Neither mapping was

significantly sensitive to Bias in separate analyses per SR-Mapping at either the start or peak of the wave (*p*s > .09).

We predicted effects in ipsilateral positivity due to preparation of the expected mapping. Paired t-tests suggested that ipsilateral positivity was enhanced in unexpected trials compared to unbiased trials for the incompatible mapping, but the enhancement was not significant either at the start of the wave ($t(13) = 2.33$, $p = .037$), or at the peak of the wave ($t(13) = 2.10$, $p = .055$). For the compatible mapping, the enhancement for unexpected compared to unbiased trials was not significant at the start of the wave ($t(13) = 1.95$, $p = .073$) nor at the peak ($p > .1$). The difference between expected and unbiased trials was negligible for both mappings in both intervals (ts < 1, $p > .1$). We expected that in unexpected trials ipsilateral positivity would be more enhanced for the compatible mapping than for the incompatible mapping, but mean amplitudes in unexpected trials were almost identical for both mappings in both intervals, confirmed by paired t-tests (ts < 1, ps > .1).

In unexpected incompatible trials, ipsilateral positivity was already apparent in an even earlier interval (see Figure 5.3, bottom-right), from as early as 350 ms prior to the response (mean RT 545 ms). In order to assess this early enhancement we analyzed ipsilateral positivity at C3/C4 between 330 and 290 ms pre-response. In this very early interval amplitudes were positive for the incompatible mapping (mean 2 $\mu V/m^2$) but negative for the compatible mapping (mean -3 $\mu V/m^2$), supported by a main effect of SR-Mapping ($F(1,13) = 13.0$, $p=.003$, $\eta_p^2 = .500$). Paired t-tests confirmed that in this interval ipsilateral positivity was enhanced for unexpected incompatible compared to unexpected compatible trials ($t(13) = 3.56$, $p = .003$), and a trend suggested that for the incompatible mapping ipsilateral positivity was enhanced in unexpected trials compared to unbiased trials ($t(13) = 2.15$, $p = .051$).

In sum, as anticipated, ipsilateral positivity was enhanced in unexpected trials, but specifically in blocks with an incompatible bias. Interestingly, ipsilateral positivity was already enhanced in an early interval for the incompatible mapping, and in unexpected trials this early ipsilateral positivity was larger for the incompatible mapping than for the compatible mapping.

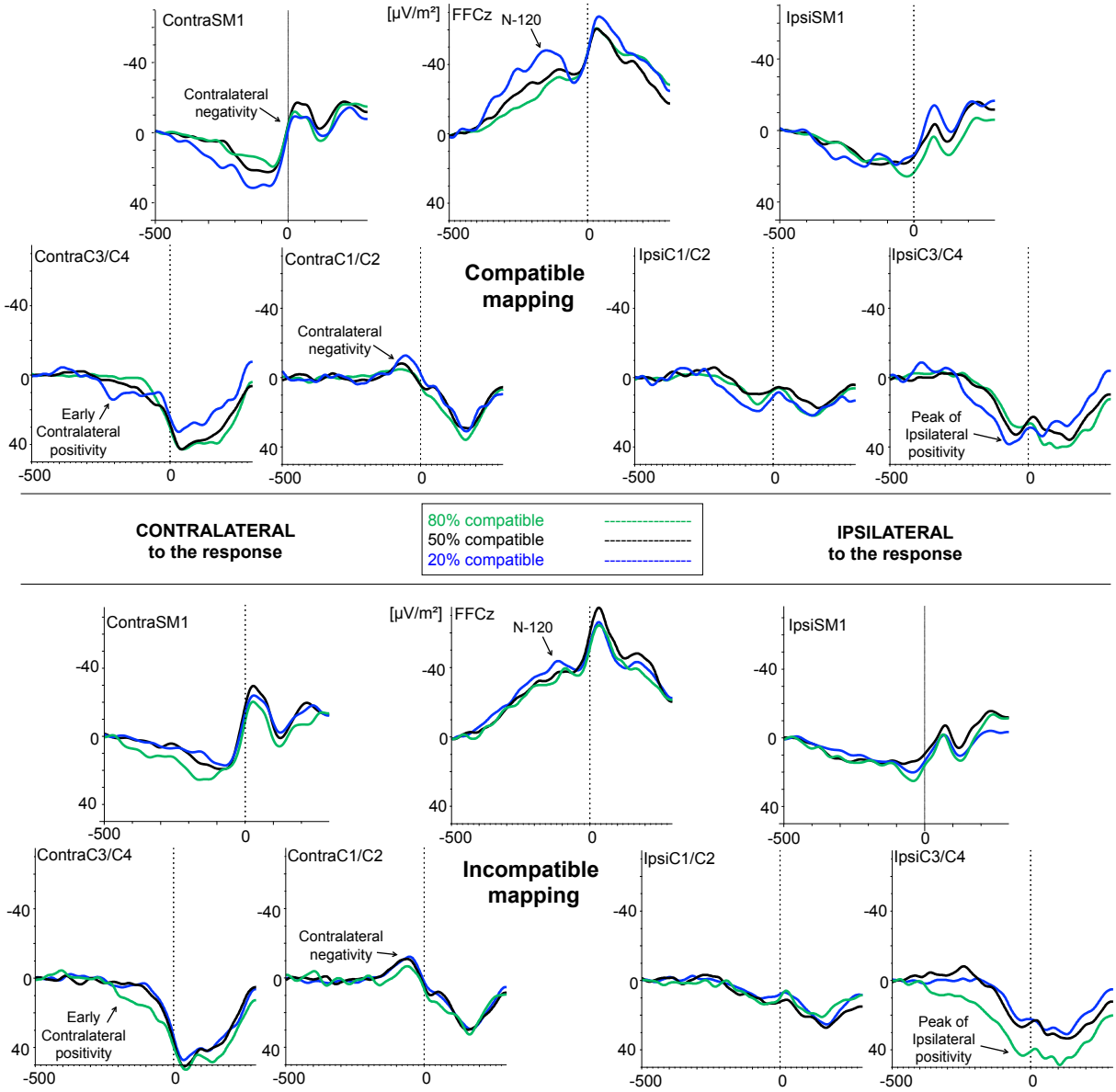

Figure 5.3. Laplacian transformations of response-locked waveforms contralateral (left) and ipsilateral (right) to the response, and midline N-120 at FFCz., for the compatible mapping (top) and the incompatible mapping (bottom). Waveforms are separated according to the bias in the block, and the baseline is the first 50 ms of the segment.

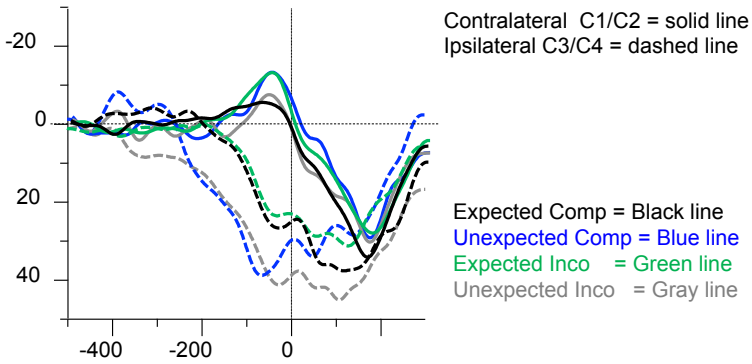

Figure 5.4. Laplacian transformations of response-locked motor preparation in the biased blocks, at contralateral C1/C2 (solid lines) and ipsilateral C3/C4 (dashed lines).

2.7.2.2 Contralateral Amplitudes

Contrary to expectation, just prior to the response (75-35 ms) amplitudes at C3/C4 were positive contralateral to the response (see Figure 5.3). However, this contralateral positivity was most apparent in unexpected incompatible trials, resulting in an interaction between Bias and SR-Mapping ($F(2,12) = 4.1$, $p=.043$, $\eta_p^2 = .408$). In separate analysis of blocks with a compatible bias, this enhanced positivity in unexpected incompatible trials resulted in a strong trend (SR-Mapping, $F(1,13) = 4.5$, $p=.053$, $\eta_p^2 = .259$), while separate analyses of blocks with no bias or an incompatible bias revealed no SR-Mapping differences (ps > .1). Neither mapping was affected by Bias, as revealed by analyses per SR-Mapping (ps > .1). Paired t-tests suggested that contralateral positivity was indeed enhanced in unexpected incompatible trials, but statistically the difference to unbiased incompatible trials was not significant ($t(13) = 2.01$, $p = .065$), nor was the difference to unexpected compatible trials ($t(13) = 1.98$, $p = .069$). No other comparisons at contralateral C3/C4 were significant (ps > .1).

Inspection of contralateral waveforms at C3/C4 suggested even earlier contralateral positivity in unexpected trials (see Figure 5.3). Therefore post hoc analyses of early contralateral positivity were performed at C3/C4 between 180 and 80 ms pre-response, the same interval as the start of ipsilateral positivity. Similar to ipsilateral positivity, contralateral positivity was slightly enhanced in unexpected trials (see Table 5.1), but the interaction between Bias and SR-Mapping was only a trend ($F(2,12) = 3.7$, $p=.058$, $\eta_p^2 = .379$). Separate analyses per SR-Mapping revealed that for the incompatible mapping early contralateral positivity was enhanced as the probability of an incompatible trial decreased (Bias, $F(2,12) = 4.1$, $p=.044$, $\eta_p^2 = .405$), but the compatible mapping was unaffected (p>.1). Paired t-tests (Bonferroni corrected) confirmed that only the difference between unbiased and unexpected incompatible trials approached significance ($t(13) = 2.63$, $p = .021$), and no other comparisons were significant (all ts < 2, all ps > .1). To verify that enhanced positivity for compatible trials wasn't in an earlier interval, we also analyzed ipsilateral C3/C4 between 230 and 200 ms and between 200 and 170 ms. However, neither ANOVAs nor paired t-tests were significant, although between 200 and 170 ms the interaction might be considered a trend ($F(2,12) = 3.3$, $p=.073$, $\eta_p^2 = .373$).

Interestingly, contralateral negativity was apparent at C1/C2 (see Figures 5.3 and 5.4, and Table 5.1), which is roughly over the area of the motor cortex that corresponds to the arm or wrist. Similar to waveforms at C3/C4 found in earlier studies, this contralateral negativity appeared to start later than ipsilateral positivity, although it peaked in roughly the same interval just prior to the response. Therefore, we analyzed contralateral negativity at C1/C2 between 75 and 35 ms pre-response. We expected contralateral negativity to be enhanced in unexpected compatible trials, in line with the hypothesis that reactive control would have to compensate for preparation of the incompatible mapping as well as decreased response activation with an incompatible bias. Contralateral negativity increased slightly as the bias changed from a compatible bias (-5 $\mu V/m^2$) to unbiased (-9 $\mu V/m^2$) to an incompatible bias (-12 $\mu V/m^2$), but the effect of Bias failed to attain significance ($p > .1$). Paired t-tests also revealed no significant enhancements for unexpected compared to unbiased trials, or for unbiased compared to expected trials, and no significant differences for SR-Mapping per Frequency (all ts < 2, all ps > .09).

In order to make a direct comparison with earlier studies, we also analyzed activation at C3'and C4'. Inspection of C3'and C4' did reveal a clear negative-going wave, which appeared to start later than found in previous studies. We tested the slope of this wave by exporting the mean amplitude at the positive peak (70-50 ms pre-response) and the mean amplitude at the negative peak (20-30 ms post-response). An ANOVA revealed no effects of Bias or SR-Mapping (all ps> .07). However, t-tests against zero revealed the negative-going wave to be significant (Bonferronni corrected) for all Bias x SR-Mapping conditions except for expected compatible-mapping trials (expected compatible, t=2.93, p=.012; all other ts>3.20, all other ps≤.007).

In sum, contralateral amplitudes were unexpectedly positive at C3/C4. An early contralateral positivity in incompatible trials was sensitive to the bias in the block, increasing in size when incompatible trials were least expected. However, contralateral negativity was present at C3'/C4', starting 60 ms prior to the response and peaking around 25 ms post-response, but it was not affected by either the Bias or the SR-Mapping.

2.7.2.3 Response-locked N-120

N-120 peaked at FFCz on average 115 ms prior to the response. N-120 latencies were analyzed by repeated measures ANOVA with the factors Bias and SR-Mapping, but no main or interaction effects of N-120 latency were significant (all *p*s>.1). The peak of N-120 was larger for compatible trials (-25 $\mu V/m^2$) than for incompatible trials (-18 $\mu V/m^2$), $F(1,13) = 5.7$, $p=.033$, $\eta_p^2 = .304$. N-120 was also enhanced with an incompatible bias (-28 $\mu V/m^2$), relative to a compatible bias (-19 $\mu V/m^2$) and no bias (-18 $\mu V/m^2$), $F(2,12) = 4.1$, $p=.044$, $\eta_p^2 = .405$. In line with the hypothesis that unexpected compatible trials would demonstrate the most prominent reactive control, N-120 was most enhanced in unexpected compatible trials (-39 $\mu V/m^2$), resulting in an interaction between Bias and SR-Mapping, $F(2,12) = 15.3$, $p<.001$, $\eta_p^2 = .718$). This interaction was supported by separate analyses of compatible trials (Bias, $F(2,12) = 16.8$, $p<.001$, $\eta_p^2 = .734$), while peak amplitudes for incompatible trials were not sensitive to Bias ($p>.1$). Separate analyses per Bias revealed a reversed mapping effect (N-120 compatible > incompatible) with an incompatible bias ($F(1,13) = 19.5$, $p=.001$, $\eta_p^2 = .600$) and with no bias ($F(1,13) = 4.7$, $p=.049$, $\eta_p^2 = .265$), but the (standard) SR-Mapping effect was not significant with a compatible bias ($p>.1$). In line with the prediction of increased reactive control in unexpected trials, paired t-tests confirmed that N-120 was enhanced in unexpected trials compared to unbiased trials for compatible trials ($t(13) = 3.69$, $p = .003$), and not for incompatible trials ($p > .1$). The enhancement for expected compared to unbiased trials was only a trend for the compatible mapping ($t(13) = 2.15$, $p = .051$) and there was no difference for the incompatible mapping ($p > .1$). In line with the prediction of increased reactive control for unprepared compatible compared to unprepared incompatible trials, comparisons per Frequency condition revealed that N-120 was indeed larger for the compatible mapping in unexpected trials ($t(13) = 2.40$, $p = .032$) and also in unbiased trials ($t(13) = 2.17$, $p = .049$). In sum, N-120 was sensitive to Bias for the compatible mapping, but not for the incompatible mapping. N-120 was largest in unexpected compatible trials, and also slightly enhanced in unbiased compatible trials.

2.8 DISCUSSION

This study examined the dynamic interplay between proactive and reactive control by manipulating SR-Mapping bias in a binary choice task. We tested the hypothesis that

reactive control would be strongest on unexpected compatible trials, rather than on unexpected incompatible trials. This hypothesis is based on two underlying assumptions concerning the interplay between proactive and reactive control. The first assumption is that proactive control prepares the expected mapping, leaving reactive control to activate the unexpected mapping when required. The second assumption is that proactive control initially reduces the level of response activation with an incompatible bias, leaving reactive control to reinstate response activation just prior to the response.

The prediction that the compatible mapping would be more sensitive to the Bias in the block was confirmed by behavioral measures Furthermore, the prediction that unexpected compatible trials would require the most reactive control was supported by the finding of an enhanced N-120 specifically in these trials. Activity recorded over the motor cortex provided support for the assumption that in unexpected trials reactive control is involved in inhibiting the incorrect response (ipsilateral positivity), and suggested that this inhibition started very early, particularly for incompatible trials. Motor activity contralateral to the response hand revealed a more complex picture, suggesting an initial inhibition rather than activation at C3/C4 (contralateral positivity), and late contralateral activation at C3'/C4' (contralateral negativity).

We expected that the complexity of the mixed SR-mapping task, in which participants are not able to predict the response hand, would result in an enhancement of both ipsilateral positivity and contralateral negativity by reactive control in unexpected trials. However, similar to the finding by Meckler et al. (2010), unexpected trials led to an enhancement in ipsilateral positivity, but not in contralateral negativity. This finding supports the hypothesis that reactive cognitive control appoints inhibition of the prepared (incorrect) response, in order to prevent error commission. On the other hand, we have found little support for the role of late activation of the unprepared (correct) response.

In line with the assumption that response activation is reduced by proactive control when expecting an incompatible trial, we hypothesized that contralateral negativity would subsequently be enhanced just prior to the response by reactive control. This assumption was based on the idea that the level of response activation can be either increased (c.f. Niemi & Näätänen, 1981) or suppressed (c.f. Band & van Boxtel, 1999; Jennings & van der Molen, 2005, Klein et al., 2014), depending on the specific strategy adopted. The absence of contralateral negativity at C3/C4 and the lateness of its onset at C3'/C4' might reflect

increased caution adopted in the current task with mixed SR-Mappings. With simpler tasks, previous studies have found contralateral negativity starting briefly following the onset of ipsilateral positivity (Meckler et al., 2010; van de Laar et al., 2012; Vidal et al., 2003). In the straight-forward choice reaction task by Meckler et al., contralateral negativity was also present but insensitive to the probability manipulation. In the current study we hypothesized that the incompatible bias would lead to an initial reduction to response activation followed by a subsequent enhancement to contralateral activation. This complements the assumptions of the DMC, which predicts enhanced proactive control when conflict is expected, as well as enhanced reactive control when conflict is detected, and exactly this interplay might result in a complex pattern of inhibition and activation to both hemispheres.

The enhanced contralateral positivity in unexpected incompatible trials is considered a manifestation of reactive control. In these trials, participants were initially expecting to give a compatible response, but needed to switch the SR-mapping following stimulus presentation. Assuming that the level of response activation is relatively higher with a compatible bias compared to an incompatible bias (c.f. Niemi & Näätänen, 1981), it might be efficient to inhibit all response activation following detection of the less frequent stimulus. Interestingly, the ipsilateral inhibition on unexpected incompatible trials was present as early as early as 300 ms prior to the response. This enhancement appeared unrelated to *reactive* control reflected in the amplitude of N-120, which peaked much later and was not enhanced for unexpected incompatible trials. In this condition, participants are expecting a compatible trial, and have to give an incompatible response unexpectedly, so it is also unlikely that this enhancement to ipsilateral positivity is related to *proactive* control. It seems that specifically on unexpected incompatible trials inhibition of the incorrect response could be triggered quickly following detection of the less frequent stimulus-feature indicating the incompatible mapping. This finding can be interpreted in terms of a Dual-Route model (e.g., De Jong, 1995; Kornblum et al., 1990; Ridderinkhof et al., 1995; Stoffels, 1996a; van Duren & Sanders, 1988), in which the direct route can be suppressed. Following the expectation of a compatible trial, suppression of the (fast) direct route would lead to inhibition of the (prepared) compatible response. In contrast, inhibition of a prepared incompatible response cannot make use of the direct route, and will therefore require more time and more cognitive control. The fast nature of the enhanced ipsilateral positivity in

unexpected incompatible trials suggests that reactive control is not necessarily dependent on conflict at the response level (Botvinick et al, 2001; Yeung et al. 2004). Instead the amount of response conflict might be efficiently predicted and prevented on the basis of stimulus features.

The expectation that enhanced proactive control would lead to the worst performance on unexpected *compatible* trials was partially, but not completely, supported. Numerically, these trials demonstrated the slowest RTs and the most errors, and the bias in the block affected the compatible mapping more than the incompatible mapping. But direct comparisons between unexpected compatible and unexpected incompatible trials revealed marginal statistical differences. However, in line with the hypotheses concerning the interplay between proactive and reactive control, unexpected *compatible* trials did involve the most reactive control (manifested in an enhanced N-120). This finding appears to contradict studies with interference tasks, in which unexpected *incongruent* trials usually seem to require the most reactive control (Bartholow et al., 2005; Gratton et al., 1992; Ridderinkhof, 2002). However, unlike Eriksen, Stroop, or Simon tasks, in the current mixed mapping task conflict is between two SR-Mappings, rather than stimulus features.

In the DMC, although proactive control is usually seen as being more effective than reactive control, proactive control can sometimes have detrimental side-effects (Braver, 2012). If proactive control prepares the expected mapping resulting in a facilitation of performance on most trials, this can still be detrimental to the minority of trials on which the unexpected mapping is required. According to the DMC, proactive control is capable of preventing conflict by maintaining task goals (Braver, 2012). In the current mixed mapping task, the most efficient performance might make use of a dynamic interplay between proactive and reactive control, whereby proactive control maintains the relevant stimulus features that indicate the mapping, rather than prematurely selecting the stimulus-response bindings. This would allow reactive control to take over following stimulus presentation.

Similar to switching between tasks, certain environments require individuals to flexibly switch between different forms of control. Research with functional Magnetic Resonance Imaging suggests that despite the ability to flexibly maintain two simultaneous control settings within one system, adaptation of cognitive control is more efficient when just one control setting is maintained (vel Grajewska et al., 2011). In other words, in some settings the amount of cognitive control needed might vary and therefore control should be

adapted accordingly. However, when different control settings are needed simultaneously (e.g. varying dependence upon proactive vs. reactive cognitive control) greater performance deficits can be expected. This offers an alternative account for the current finding that rare compatible trials were associated with the worst performance. Assuming that proactive control dominates in blocks with an incompatible bias (c.f. Braver, 2012), applying appropriate reactive control in unexpected compatible trials will take more effort. On the other hand, assuming that in blocks with a compatible bias reactive control dominates, reacting to unexpected incompatible trials will require relatively less effort.

We suspect that successful performance in the current tasks can best be accounted for by an efficient collaboration between proactive and reactive control. For example, when expecting an incompatible trial, if participants have employed the strategy (proactively) of maintaining a low level of response activation (c.f. Niemi & Näätänen, 1981), then less reactive control is needed to inhibit the prepared incompatible response. This account of the relationship between proactive and reactive control also complements the finding that successful training of cognitive control leads to a shift from more *reactive* control to more *proactive* control (Berkman et al., 2014; Braver et al., 2009). However, it should be noted that not all forms of increased proactive control are necessarily efficient. In the current task, the large amount of errors in unexpected compatible trials appears to be related to too much preparation of the incompatible response and too little use of reactive control to correct that preparation. In other words, the proactive strategy employed by some participants appears to be efficient only for expected trials. Even if participants have proactively reduced the response activation level (perhaps the most successful strategy when expecting an incompatible trial), theoretically this implies that more reactive control will be needed to activate the compatible response (contralateral negativity). Unfortunately the current experiment could not clarify the role of contralateral negativity in reactive control. Future research might be able to untangle the relationship between different proactive strategies and late activation of the correct response (contralateral negativity), for example using simpler SR-bindings but with differing task instructions.

The current study confirms N-120 as a useful electrophysiological measure of reactive cognitive control. The functions underlying N-120 are still not clear, and relating N-120 to other electrophysiological measures is made particularly difficult as this measure partially reflects the positive component at the response. Considering that N-120 was specifically

enhanced for unexpected compatible trials, in which the most errors were committed, we assume that the control reflected in the amplitude of N-120 is sensitive to response conflict (c.f. Botvinick et al, 2001; Yeung et al. 2004). This interpretation also complements the finding by Lungu et al. (2007) of increased activation of ACC in unexpected trials. Similar to the interpretation of EMG-locked N-40 (Meckler et al., 2010), we also suspect that N-120 reflects one agent of ipsilateral positivity, with specific influence around the peak of ipsilateral positivity (75-35 ms). In other words, reactive control is one, but certainly not the only agent of inhibition. Previous studies have linked inhibition to preparatory (proactive) control (e.g. Band & van Boxtel, 1999; Band et al., 2003; Jennings & van der Molen, 2005; Klein et al., 2014), or to subliminal primes (Praamstra & Seiss, 2005). As discussed above, it is also likely that inhibition of the incorrect response can often be triggered by an efficient collaboration between proactive and reactive control, such that conflict at the response level can be prevented . In the current study, it appears that response conflict is prevented in unexpected incompatible trials, but not in unexpected compatible trials. The current study was unable to elucidate the role of contralateral negativity, partly because amplitudes at C3/C4 were positive rather than negative (suggesting partial inhibition of the correct response), and partly because differences between conditions at C1/C2 were not significant. We expect that late activation of the correct response by reactive control is related to the strategy employed by proactive control, and future research might be able to unravel the nature of this relationship.

2.9 CONCLUSIONS

The current study revealed an interplay between proactive and reactive cognitive control, such that in some circumstances a proactive control strategy could be detrimental to performance. The electrophysiological measures support the hypothesis that ipsilateral inhibition is one of the mechanisms that can be appointed by cognitive control, be it proactive or reactive control. We suspect that specifically late reactive control (reflected in the amplitude of N-120) is influenced by short-term response conflict. However, the best strategy does not rely upon only late reactive control, but more likely upon a successful collaboration. Ideally, proactive control might prepare for efficient selection of the relevant stimulus features needed to engage efficient reactive control.

Chapter 6 (Discussion):
General and Specific Mechanisms of Cognitive Control

This chapter aims to relate the findings of the four experimental chapters to the initial goals of the project and this thesis - to gain insight into the flexibility of cognitive control - by discussing the research at three conceptual levels. Level 1 of the discussion relates to individual experiments, presenting a concise summary of the findings from each experimental chapter. Level 2 of the discussion addresses each of the major research questions presented in the general introduction, relating them to the findings from the experimental chapters and the surrounding literature. Finally, by including more recent developments in research on the role of cognitive control, discussion Level 3 aims to integrate the research questions, to provide insight into the flexibility of control, to consider the limitations of this thesis and unanswered questions, and to outline potential directions for future research.

6.1 Summary of Experimental Findings

Chapter 2 assessed response conflict and online cognitive control with two types of interference: Simon (location) and Eriksen (flankers). We found support that Simon and Eriksen interference reflect different sources of conflict when considered in the context of a dual-route model of SRC: larger, later interference effects for Eriksen compared to Simon interference in performance and motor preparation; and an N350 at midline and contralateral central electrodes that was only present with incongruent flankers but not locations. However, we could not completely rule out the possibility that differences between Eriksen/Simon interference reflected only a difference in the time taken to process locations or flankers via the direct route, i.e., a temporal account. Furthermore, the lack of Simon effects at midline components made it difficult to determine which online control mechanisms might have been involved in resolving Simon interference. Potentially, the decision to present Eriksen and Simon interference in separate experimental blocks meant that response conflict was prevented on some trials or resolved quickly on other trials by preparatory strategies that acted on the predictability of specific types of interference. Chapter 3 aimed to disentangle these ambiguities.

Chapter 3 elaborated on the findings reported in chapter 2 in favor of the idea that cognitive control resolves response conflict in a domain-specific manner. By presenting Eriksen and Simon interference in the same experimental blocks, and comparing both compatible and incompatible SR-mappings in performance and ERPs, we were able to identify independent sources of response conflict and to some extent independent mechanisms of control for each type of interference. Reflecting differing degrees of dimensional overlap for each type of irrelevant interference (flankers/location) with either the relevant stimulus attribute (arrow direction) or the relevant response dimension (left/right hand), Simon and Eriksen interference demonstrated a different pattern of response conflict with compatible and incompatible SR-mapping tasks: congruency effects between target arrow and location reversed, such that irrelevant stimulus locations consistently facilitated the corresponding response; while congruency effects between target and flanker arrows were reduced but not reversed.

With a compatible mapping, we replicated the finding in chapter 2 of larger interference effects for Eriksen compared to Simon interference in response times (RTs), but effects on error proportions and motor preparation were similar with each type of interference. With a reversed SR-mapping, Simon effects appeared to reverse such that irrelevant locations still facilitated the response corresponding to the stimulus side (in RTs, errors and motor preparation), rather than demonstrating a reversed SR-mapping rule (e.g. left=right). In contrast, in incompatible blocks, Eriksen effects were reduced in RTs, and eliminated in errors and motor preparation. An elimination of Eriksen interference with the reversed mapping is somewhat in favor of traditional accounts, such that incongruent flankers partially activate the spatially corresponding response via direct route priming, but not to the same degree as an irrelevant stimulus location, suggesting a combination of direct and indirect route effects. Midline N1 and N2 were enhanced for all Simon stimuli (congruent and incongruent) compared to Eriksen stimuli, suggesting increased online control with interference by locations compared to flankers. In line with the Dual Mechanisms of Control model (Braver et al., 2007), we interpreted the combined N1-N2 finding to reflect a strategy in mixed interference blocks that prepares for the worst (incongruent flankers), but uses reactive control to quickly discriminate unilateral stimuli as a potential source of interference requiring voluntary suppression of the direct route.

Chapter 4 assessed response strategies in more detail by comparing experimental blocks in which the task (SR-mapping) was either blocked or mixed. With a blocked presentation, we assumed that preparatory (proactive) control should be able to set up the SR-bindings in advance, thereby minimizing response conflict and the effort required for online (reactive) control to resolve interference. With a mixed presentation, in which the SR-mapping (compatible vs. incompatible) was unpredictable, we assumed that proactive control would be unable to resolve response conflict as efficiently as was possible with a blocked SR-mapping, particularly when switching between SR-mappings, resulting in enhanced measures of response conflict and reactive cognitive control.

As predicted, in mixed task blocks performance was significantly worse and SR-mapping effects were eliminated, reflected in an enhanced stimulus-locked N2 in all mixed conditions. Mixed incompatible trials demonstrated preferential response activation in favor of the incorrect response in LRPs, but mixed compatible trials demonstrated the greatest degree of response conflict (reflected in errors and CRN) and enhanced reactive cognitive control (reflected in a response-locked mid frontal N-120). Furthermore, sequential effects analysis revealed that N-120 was specifically enhanced when switching from an incompatible to a compatible SR-mapping, and paired with an enhancement to the peak of the response-locked LRP, suggesting that this component reflects correction of preferential response activation. We concluded that the enhanced N2 in all mixed tasks reflected increased demands on proactive control to select the appropriate SR-mapping, possibly involving inhibition of the inappropriate SR-mapping; whereas enhanced N-120 reflects late correction when inadequate preparation of the SR-mapping leads to additional response conflict. Chapter 5 assessed the effects of incorrect task preparation on N-120 in more detail.

Chapter 5 investigated the determinants and the dynamics of online (reactive) cognitive control depending on the degree to which SR-bindings (the task) can be predicted prior to stimulus presentation. We used high-density EEG, which allowed us to calculate Laplacian transformed ERPs over mid frontal cortex and motor cortex, in order to assess the extent to which proactive and reactive control mechanisms influence the level of response activation contralateral and ipsilateral to the response. We manipulated the predictability of the SR-mapping by varying the probability of compatible vs. incompatible SR-mappings (80/20, 50/50, 20/80). We predicted that participants prepare for the expected SR-mapping, such

that on unexpected trials reactive control is needed to correct the SR-mapping, by inhibiting the incorrect response tendency, reflected in contralateral negativity and ipsilateral positivity.

In line with previous studies, SR-mapping effects depended upon the bias in each experimental block, such that with a compatible bias, participants performed worse with an incompatible mapping, but this effect was eliminated with no bias, and reversed with an incompatible bias. Reactive control (reflected in response-locked N-120) was enhanced with unexpected compatible trials, in line with the finding reported in chapter 4 that correcting SR-bindings after preparation of the incompatible mapping requires the greatest degree of online control. However, ipsilateral positivity between N-120 and response execution was enhanced for all unexpected trials, suggesting that inhibition of the incorrect response tendency might be recruited by reactive control reflected in N-120 or even earlier. Reactive control appears to reflect both the extent to which SR-bindings have been prepared in advance as well as the difficulty of the tasks – such that more control is needed when switching from a difficult to an easier task. Presumably, the dynamic interplay between proactive and reactive control needed during performance of mixed tasks led to complex patterns of response conflict, which could be assessed further using both high density EEG and computational modeling.

6.2 Addressing the Major Research Questions

This part of the discussion addresses the individual research questions in turn, so that they can be integrated in the final level of the discussion to provide a foundation for an analysis of the overall flexibility of cognitive control. Each of the major research questions is divided into logical sub-questions before offering an interim conclusion in answer to the research question. To recall, the major research questions are:

1. What are the mechanisms of online reactive control processes involved in resolving unexpected interference between competing responses, and can we use ERPs to differentiate the neural correlates of conflict from functional control mechanisms?
2. To what extent can stimulus-response bindings be prepared in advance to allow automatic responding upon presentation of the stimulus?

3. Are the mechanisms of conflict resolution domain general or domain specific, and which control strategies are most successful with specific types of conflict?

It is not a simple to task to address each of these questions exclusively, particularly considering the hypothesized interactions between preparatory and online reactive control, such that the former might eliminate the requirement for the latter. While it is feasible that sufficient preparation for all expected stimuli, and subsequent practice at the task, might reduce the need for online control, it is difficult to prove that updating or strengthening SR-bindings can occur without online adjustments to cognitive control at all (implying interpretation of null effects). Determining the extent to which this is possible would benefit from a definitive answer to the first question in this thesis, by identifying the mechanisms of online/reactive control. Defining online reactive control also seems less of a challenge than defining preparatory control (but see Jennings & van der Molen, 2005, for a thorough review and definition of multiple features of preparation for action), due to the assumed temporal (stimulus to response) and functional (modulated by response conflict) attributes of reactive control. As such, I will first address the insights from this thesis on the mechanisms of online, reactive control in response to conflict (research question 1), and subsequently use these insights to consider the extent to which the decision can be pre-programmed by proactive control (research question 2). The final question, regarding the domain-specificity of control, is probably the most difficult to answer, and therefore will benefit from any insight into questions 1 and 2, as well as from recent work by others trying to assess the independence of control mechanisms.

6.2.1 Research Question 1: Online Control and Conflict

What are the mechanisms of online reactive control involved in resolving unexpected interference between competing responses, and can we use ERPs to differentiate the neural correlates of response conflict from those that reflect functional control mechanisms?

In order to elucidate the essential mechanism(s) of reactive online cognitive control, I will first describe interference and its resolution in terms of conflict and control. Subsequently, the discussion will address interference from decision-*relevant* vs. -*irrelevant* stimulus features separately, before finally integrating these to define reactive online control mechanisms in relation to the ERP measures.

6.2.1.1 Conflict vs. Control

In all four experimental chapters, we used SRC to manipulate the amount of interference, and found negative midline frontal components associated with resolution of each type of interference. However, we noted with every study that such N2-like midline components are open to competing interpretations: do these components reflect functional cognitive control processes involved in the resolution of interference, or the monitoring or detection of (response) conflict? Furthermore, assuming that adjustments to cognitive control require detection of conflict (Botvinick et al., 2001), then what exactly is conflict? Botvinick and colleagues (2001) assumed that conflict is detected by anterior cingulate cortex (ACC), and reviewed many studies that had identified increased ACC activation in conditions that are typically associated with competition between responses. There is abundant evidence that ACC, or medial frontal cortex (MFC), is activated by response errors, response conflict, decision uncertainty, and even by unfavorable outcomes (see Ridderinkhof et al., 2004, for a review), supporting the common assumption that MFC (and ACC) play a role in performance monitoring (Botvinick et al., 2007; Ridderinkhof et al., 2004). Botvinick et al. (2001) and others (e.g. Carter & van Veen; Yeung et al., 2004) have used simulations to mimic typical interference tasks, incorporating a feedback loop from a conflict monitor to control over task goals, which predict the degree of conflict depending on the sequence of trials and subsequent adjustments to cognitive control between trials. According to these models, the degree of conflict detected is perfectly correlated with the subsequent increase in cognitive control.

Rather than relying upon simulations, the studies presented in this thesis attempted to distinguish the degree of conflict from the amount of control by assessing motor preparation. Due to the requirement to decide between a left vs. right hand response in all tasks, we reasoned that, if midline N2 reflects the detection of conflict between competing responses, N2 should be enhanced when conflict is also reflected in errors and motor preparation. After assessing performance to identify the trial-types with the most conflict, we defined the temporal dynamics of response competition in motor cortex activation as an interval in which preferential response activation suggested relatively reduced activation of the correct response compared to comparable low conflict trials. In each chapter, response competition was assessed using slightly different measures, depending on the effects of

interest and number of electrodes available, but across tasks we found a clear relationship in high-conflict trials between measures of performance and motor preparation. An important differentiation between the types of conflict manipulated, which undoubtedly influences how mechanisms of control are engaged, concerns whether the stimulus attribute causing the interference needs to be processed to determine the response decision (as with the mapping effect) or not (as with Simon/Eriksen effects). Accordingly, decision-relevant and -irrelevant stimulus features will first be considered separately.

6.2.1.2 Conflict Resolution with Interference by Irrelevant Features

We assessed response conflict and its resolution with task-irrelevant Eriksen and Simon interference in chapters 2 and 3, measuring individual contributions of motor preparation at C3/C4 contralateral and ipsilateral to the response (c.f. Yordanova et al., 2004), and found that high conflict conditions were associated with reduced negativity contralateral to the response around the timing of N2. Although the exact interval of response conflict appeared to depend upon both the type of interference and the experiment (i.e., size of the stimulus-set), this was somewhere between 220-330 ms following stimulus onset for Eriksen interference, and notably inconsistent between studies for Simon interference.

With *Eriksen interference*, control mechanisms appeared to depend upon the stimulus set, potentially accountable to the predictability of flanker interference. When Eriksen trials were presented in separate experimental blocks to Simon trials (chapter 2), a midline and contralateral N350 was enhanced with incongruent flankers. But when Eriksen interference was mixed with Simon interference, neither the late midline N2 (around 300 ms post-stimulus) nor contralateral N300 were significantly enhanced with incongruent flankers relative to congruent flankers. In fact, reduced negativity at contralateral N300 with incongruent flankers suggested that response conflict was unresolved even at 300-330 ms post-stimulus. Importantly, only with blocked Eriksen interference was a late N2 (N350) enhanced for high conflict trials, suggesting that, unlike motor preparation, N2 did not consistently reflect the degree of response conflict. Yet there were similarities in N2 with Eriksen interference between chapters 2 and 3 -at both contralateral and ipsilateral motor areas, N2 appeared to be wider with incongruent flankers, suggesting a later component specific to resolving flanker-related response conflict.

Previous Eriksen studies using letter stimuli found enhanced midline N2 with incongruent flankers. That is, around 340-380 ms following presentation of the target in the study by van Veen & Carter (2002), which presented flankers 100 ms prior to the target; and around 250-350 ms specifically in blocks in which incongruent flankers were 80% probable in a study by Bartholow et al. (2005). Kopp et al. (1996) used arrow stimuli and presented flankers 100ms earlier than targets, and found a clear enhancement to N2 with incongruent flankers at around 300ms post-target. Compared to these earlier studies, Eriksen effects on midline N2 were similar to van Veen & Carter (2002) in chapter 2, but absent in chapter 3, despite the presence of response conflict reflected in motor preparation. Potentially then, presenting mixed interference led participants to proactively inhibit processing of irrelevant flankers, such that Eriksen interference by arrow stimuli could be resolved without detection of response conflict.

Response conflict with *Simon interference* (chapters 2 and 3) was also largely dependent upon the design: blocked vs. mixed interference. The very early deflections with Simon stimuli in both studies subsided prior to the N2 measurement intervals and in chapter 2 this deflection was maximal at posterior channels, suggesting contamination of early motor activity (e.g. Valle-Inclan, 1996). However, a relevant contradiction between chapters 2 and 3 is the absence of Simon effects at contralateral N2 in chapter 2 (blocked interference), yet a clear interval of reduced contralateral negativity in chapter 3 (mixed interference) from 300-330 ms post stimulus. Similar to the interpretation of Eriksen interference in chapter 3, it is possible that with blocked interference Simon effects were largely resolved by preparatory control. Most interesting is the finding that with mixed interference reactive control reflected in both midline N1 and N2 was enhanced for all Simon stimuli (and to a lesser extent neutral trials), but independent of any conflict resulting from either congruency or the mapping effect. As such, midline N2 was not sensitive to response conflict, but possibly to the prediction of potential response conflict, which was apparent at contralateral N300 in high-conflict trials, just after the peak of midline N2. Due to the unpredictability of conflict and even the type of interference (Eriksen/Simon), a useful strategy would be to suppress the direct route prior to detection of conflict, but then only for Simon interference, which takes effect via the direct route.

From this perspective, both the enhanced midline N1 and N2 with Simon stimuli could be interpreted as electrophysiological concomitants of reactive within-trial control

that responds to specific stimulus features defined by proactive control. One recent model of cognitive control adjustments can account for this interpretation (Scherbaum et al., 2012), even without assuming a role for conflict monitoring. In their model, Scherbaum et al. (2012) included direct bottom-up connections from an input (stimulus) layer to the goal layer (cf. Gilbert & Shallice, 2002), such that salient stimulus attributes are able to influence the strength of current task goals, thereby accounting for within- and between trial performance effects, as well as global effects such as the probability of conflict trials. Although many studies, including some of those in this thesis, suggest a role for detection of response conflict, it is insightful to consider that control adjustments can also be accounted for without explicit inclusion of conflict monitoring.

6.2.1.3 Conflict Resolution with Interference by Relevant Features (Mapping Effects)

We assessed resolution of response conflict incurred with the increased cognitive load of mixed SR-mappings, including task switching, in chapters 4 and 5. In chapter 4 we detected the presence of preferential response activation in the LRP using t-tests against zero for difference waves (c.f. Guthrie & Buchwald, 1991); and in chapter 5 we assessed Laplacian transformed signals over motor cortices. Preferential incorrect response activation was only statistically significant in mixed incompatible SR-mapping trials (chapter 4) from 258 to 352 ms, but this interval was longer when preceded by compatible trials and shorter when preceded by incompatible trials, which is in line with the additional time needed when switching between tasks (see Monsell, 2003, for a review of task switching costs). Stimulus-locked N2 was enhanced for all mixed compared to blocked tasks (SR-mappings), shedding doubt on the hypothesis that this component was sensitive to the trials with the most conflict.

Interestingly, it was mixed *compatible* trials that demonstrated the most errors, specifically when preceded by incompatible trials, paired with an enhanced midline response-locked N-120. The association between errors and N-120 for compatible 'switch' trials might mean that N-120 reflects the detection of response conflict, but considering that these were correct response trials, N-120 could also reflect within-trial control adjustments that correct response preparation (c.f. Burle et al., 2002; Ridderinkhof, 2002a). Rather than incorrect preferential response activation, mixed compatible trials demonstrated early activation toward the correct response (only reliable when preceded by compatible trials)

that subsided at around 350 ms before the onset of final response preparation after 400 ms. Without evidence of response conflict in motor preparation prior to N-120, it is unclear whether N-120 reflected conflict detection, and measuring response conflict in mixed tasks clearly cannot rely upon LRP measures. Crucially, the finding that both errors and N-120 were increased more when switching to the compatible mapping than when switching to an incompatible mapping favors a strategy whereby participants prepared for an incompatible mapping, so it seems more efficient to monitor the relevant cue for the SR-mapping rule (gaze eccentricity in chapter 4), than to wait for the detection of response conflict.

Using high-density EEG in chapter 5, we looked in more detail at how online control corrects the SR-mapping rule in unexpected conditions, using response-locked Laplacian transformed signals. The trials that resulted in the slowest RTs and the worst errors were rare compatible trials, and again these trials were paired with an enhanced N-120. Notably, these unexpected compatible mapping trials initially demonstrated enhanced positivity *contralateral* to the response, followed later by enhanced positivity *ipsilateral* to the response, which might be interpreted as early inhibition of the correct response and later inhibition of the incorrect response (c.f. Burle et al., 2004). This demonstrates how sufficient spatial resolution can help to identify conflict and it's resolution even with mixed tasks (SR-mappings), building on previous assessments of motor preparation in simpler tasks (e.g. Burle et al, 2004; Carbonnell et al., 2004; Meckler et al., 2010). Together, the data from chapters 4 and 5 reveal that reactive control was needed most when switching from an incompatible to a compatible SR-mapping. There is little doubt that in these mixed SR-mapping tasks participants were biased toward preparing for the more difficult incompatible mapping, but it is still open to discussion whether the N-120 enhancement was modulated by detection of response conflict, detection of an incorrect SR-mapping, or even mechanisms of control involved in correcting the SR-mapping without conflict detection.

6.2.1.4 Interim Conclusion 1: What are the mechanisms of online reactive control?

All four experimental studies revealed mechanisms of reactive control that were modulated by unpredictable conditions, potentially or certainly associated with response conflict. In left/right hand decision-making tasks, it seems that the most sensitive ERP measures of response conflict assess separate contributions of motor preparation contralateral and ipsilateral to the response. Specifically, the presence of response conflict can be associated

with reduced negativity or increased positivity contralateral to the response, and conflict resolution can be associated with later increased negativity *contralateral* to the response (chapter 2) or increased positivity *ipsilateral* to the response (chapter 5), the latter depending on the spatial resolution of the signal (c.f. Vidal et al., 2003).

The stimulus-locked fronto-central midline components in each task did not consistently reflect the degree of response conflict apparent in performance or motor preparation. Particularly the results of chapters 3 and 4 support the idea that stimulus-locked N2 reflects more than the detection of conflict, potentially even strategic (proactive) control mechanisms involved in preventing response conflict. On the other hand, stimulus-locked N350 with incongruent flankers (chapter 2) and the response-locked N-120 (chapters 4 and 5) appear to be specifically modulated by response conflict. Others have found that N2 latency correlates positively with response times (e.g. Gajewski et al., 2008; Nieuwenhuis et al., 2003), which has led to the suggestion that an N2 time-locked to the stimulus reflects detection of response conflict, while an N2 time-locked to the response is more likely to reflect the mechanisms associated with resolving conflict (Nieuwenhuis et al., 2003). However, in mixed tasks we found that response-locked N-120 and stimulus-locked N2 could be functionally and temporally differentiated into two separate components, and only N-120 was enhanced for the trials demonstrating the most conflict (chapter 4). To some extent the difficulty of the mixed SR-mapping task must have introduced additional processing, delaying the onset or detection of response conflict, but an alternative interpretation is that neither of these components are specifically related to conflict detection processes, and both reflect online control that strengthens the SR-bindings associated with the relevant task goals. A similar interpretation could be given to N2 with Simon interference in chapter 3, in line with the idea that salient stimulus attributes can trigger online adjustments to the strength of task goals (c.f. Scherbaum et al., 2012). In the case of Simon interference (or other types of interference that induce direct response activation), the task goals might include suppression of the direct route.

6.2.2 Research Question 2: How Effective is Preparatory Control Alone?

To what extent can stimulus-response bindings be prepared in advance to allow automatic responding upon presentation of the stimulus?

This research question essentially harks back to the extent to which decision-making might be accounted for without online (reactive) cognitive control, as well as contemplating the potential mechanisms of proactive control. As such, the discussion will first address conditions in which preparatory control might be sufficient, in light of the findings with Simon and Eriksen interference, and subsequently address conditions in which resolution of response interference appeared to depend upon reactive control, such as when encountering an unexpected SR-mapping. Finally, in answer to the research question, the limits of preparatory control are outlined.

Throughout the studies in this thesis, we have assumed that resolving unexpected interference reflects within-trial control adjustments (c.f. Burle et al., 2002; Ridderinkhof, 2002a), and that a complete account of decision-making in SRC tasks requires a combination of proactive and reactive control in order to perform a task accurately (c.f. Braver et al., 2007; Braver, 2012; Ridderinkhof, 2002a). Alternatively, perhaps control is primarily preparatory, in line with the idea of a "prepared reflex" (Exner, 1879; Hommel, 2000), and differences in response times reflect only the time needed to activate the correct response, which in turn depends on the precision with which task goals (and associated SR-bindings) have been set up in advance. By this view, with practice at the task, all relevant SR-bindings would be strengthened *automatically*, so there might not be any need to increase cognitive control either within or between trials.

6.2.2.1 Conditions that Require Little Online Control

Many of the findings presented in this thesis suggest that online control does not consistently relate to response conflict, so response conflict might be resolved without reactive control adjustments. With Simon interference in chapter 2, response conflict reflected in ERPs appeared to be resolved before the peak of midline and contralateral N2, shedding doubt on the need for online control with interference. In chapter 3, measures assumed to reflect enhanced control (midline N1and N2) depended upon the type of unpredictable interference (Simon vs. Eriksen), but were not modulated by response conflict. With Eriksen interference in chapters 2 and 3, we found evidence of response conflict reflected at contralateral N2, yet no enhancement to the peak of midline N2 with mixed interference (chapter 3), and a delayed congruency effect (in N350) with blocked interference (chapter 2). Considering the larger and later effects of Eriksen compared to

Simon interference at contralateral N2 (chapter 2), it is perhaps not surprising that N2 with incongruent flankers was visibly wider in grand averaged ERPs, demonstrating greater variability. In line with findings that N2 latency correlates with response times (e.g. Gajewski et al., 2008; Nieuwenhuis et al., 2003), the slower RTs, response conflict reflected at contralateral al N2, and the wider (but not enhanced) N2 with Eriksen interference implies a general response slowing and associated increase in variability, but not necessarily reactive online control.

Such a response slowing might be interpreted either as additional time needed to activate the correct response (c.f. Hommel, 2000), or as a general reduction in response activation (c.f. Band & van Boxtel, 1999; Band et al., 2003; Jennings & van der Molen, 2005). Neither of these accounts requires the inclusion of mechanisms of online/reactive cognitive control that resolve response conflict within a trial (c.f. Braver, 2012; Burle et al., 2002; Ridderinkhof, 2002a), so potentially Eriksen interference can be resolved by proactive control alone. Although N350 (chapter 2) was associated with incongruent flankers, this component reflected an enhancement to negativity over motor areas contralateral to the response, which could reflect delayed response selection in a large proportion of trials/participants, even without adjustments to reactive control. At least in tasks in which the SR-mappings are blocked, such as in typical Eriksen and Simon tasks, sufficient preparation might eliminate the need for reactive control adjustments.

6.2.2.2 Conditions that Require More Online Control

However, the findings with mixed SR-mappings presented in chapters 4 and 5 are more difficult to interpret without including a role for reactive online control. The response-locked N-120 was associated with the trials that demonstrated the most conflict in accuracy measures: compatible trials that were unexpected (chapter 5) or followed incompatible trials (chapter 4). Crucially, a raster-like plot (chapter 4) revealed that stimulus-locked N2 and response-locked N-120 could be differentiated temporally as well as functionally, and N-120 was also paired with a late enhancement to preferential response activation in favor of the correct response. In other words, although N-120 varied visibly with response-times, this component was not a delayed N2, and reflected different mechanisms than those reflected at N2.

The dissociation between N2 and N-120 is difficult to account for without assuming a role for reactive control processes that correct or at least strengthen SR-bindings within a trial. However, there are alternative accounts whereby N-120 might be a correlate of control that takes effect between-trials rather than within-trials. For example, if we assume that stimulus-locked N2 reflects the proactive maintenance of all SR-bindings in working memory, then in line with one potential role for reactive control (Braver, 2007), as well as the idea of micro-adjustments to task-set (Ridderinkhof, 2002b), N-120 might reflect conflict-driven reactive/micro adjustments to those global task goals. This implies that such control could be reactive to competition between responses, but result in a proactive advantage on the following trial. Such an interpretation of N-120 is in line with conflict-monitoring theories (e.g. Botvinick et al., 2001; Carter & van Veen; Yeung et al., 2004), which assume that detection of conflict on one trial leads to a subsequent increase in control that facilitates performance on the following trial, but by-passes the potential role of within-trial control adjustments (Braver et al., 2007; 2012; Burle et al., 2002; Ridderinkhof, 2002a).

However, in support of both within-trial and between-trial control adjustments, the post-response CRN (the negative component that follows correct responses) represents a more likely manifestation of between-trial micro adjustments to task set than the pre-response N-120. Extending on previous interpretations of CRN as detection of an inappropriate response strategy (Bartholow et al, 2005), this interpretation makes sense because our results suggested a strategic bias toward preparing for an incompatible SR-mapping with mixed interference, and CRN was enhanced following all mixed compatible trials, suggesting that CRN reflects a strengthening to this bias following compatible trials. Ridderinkhof (2002b) suggested that micro adjustments to task set reflect the degree of cautiousness, and demonstrated that such cautiousness increases following errors, which are consistently associated with an ERN/Ne (e.g. Falkenstein et al., 1991; Gehring et al., 1993). Importantly, Ridderinkhof (2002b) found that errors resulted not only in post-error-slowing (c.f. Rabbit & Vyas, 1970), but also in benefits to incongruent trials, but only when incongruent trials were less probable (25%), supporting the suggestion that micro adjustments affect the relative amount of direct response activation vs. suppression (Ridderinkhof, 2002a, 2002b). While such micro effects (e.g. as reflected in CRN in chapter 4) are reactive in the sense that they are triggered by response conflict, the effect that they have on subsequent trials is more proactive in nature, in anticipation of subsequent conflict.

Nevertheless, the findings in relation to reactive control reflected at both N-120 and CRN reveal conditions in which control seems to require adjustments both within- and between-trials, contradicting the extreme standpoint of the "prepared reflex" (Exner, 1879; Hommel, 2000).

6.2.2.3 Interim Conclusion 2: The Limits of Preparatory Control

Taken together, the findings presented in this thesis are compatible with a dynamic interplay between proactive and reactive control (c.f. Braver et al., 2007). Proactive control might be sufficient, almost to the extent of a prepared reflex, when the task demands are low or sources of potential conflict are predictable (e.g. with Simon stimuli in chapter 2 and with the blocked incompatible mapping task in chapter 4); but when interference is less predictable or unexpected, reactive control will be needed to suppress (e.g. Simon interference in chapter 3) or to correct (e.g. incorrectly prepared compatible mapping trials in chapters 4 and 5) response activation within a trial, as well as between trials (e.g. following compatible trials in chapter 4). Potentially, reactive control is most essential during the learning or early phase of a task (particularly in an experimental task setting), but its role is gradually eliminated by practice. Future studies could investigate the role of practice in complex tasks such as the mixed SR-mapping task on reactive control components, to assess whether in high conflict trials errors and reactive control components such as N-120 reduce with practice, even when associated interference effects are still robust in RTs.

Finally, one complexity when the probability of each task (or stimulus) is manipulated (chapter 5) is dissociating between effects of expectancy vs. effects of practice. However, with unpredictable mixed SR-mappings in chapter 4, there was no difference in the probability of each SR-mapping, supporting the idea that the bias toward the incompatible mapping reflects top-down preparatory control over task goals (e.g. Jennings et al., 2002; Jennings & van der Molen, 2005; Verbruggen, 2016). If we assume that preparatory processes can configure SR-bindings similar to a prepared reflex, then this implies that a decision can be both intentional and automatic, eliminating the boundary between these otherwise separate concepts (see Hommel, 2000; Ridderinkhof, 2014; Verbruggen, 2016). Although this argument is highly appealing, supporting such an account is beyond the scope of this thesis, and greater potential lies in using computational modeling to simulate both behavioral and neurophysiological data in blocks of conflict tasks without incorporating

feedback loops or top-down control that initiates online control adjustments. In sum, the effectiveness of proactive control should be highly dependent on specific (proactive) strategies, which will be discussed in more detail in answering the final research question in this thesis, but also on the motivation and abilities of the individual (Braver, 2012).

6.2.3 Research Question 3: Is Cognitive Control Domain-Specific?

Are the mechanisms of conflict resolution domain general or domain specific, and which control strategies are most successful with specific types of conflict?

Answering this final research question requires first defining the potential sources of interference. Subsequently, studies that have used 'congruency sequence effects' (CSEs) to assess the domain-specificity of control adjustments between-trial will be presented, before turning to the focus of this thesis on a critical analysis of ERP measures that offer comparison of within-trial control with different sources of interference. Finally, the discussion considers the extent to which control strategies might be either specific to a particular source of interference, specific depending upon other factors such as task difficulty, whether different strategies might be employed flexibly, or whether there might in fact be a general mechanism that can account for the multiple effects of control adjustments.

In order to assess whether different forms of conflict are resolved independently (domain-specific), it is paramount to ensure that the two types of conflict are independent of each other (see Egner, 2008; Braem et al., 2014). Making use of the fundamental principles of the Dimensional Overlap Model (Kornblum et al., 1990), there are at least three potential sources of response conflict that have been compared and described in this thesis. The first is interference between relevant response and irrelevant stimulus dimensions (Simon), the second is interference between relevant and irrelevant stimulus dimensions (Eriksen), and the third is interference between relevant stimulus and relevant response dimensions (mapping effects). Although each of these types of interference is assumed to accumulate at the response level (with the exception of Eriksen neutral trials, which represent only stimulus conflict), the path of the interfering stimulus attribute is different in each case. The assumption and findings in this thesis support the idea that traditional Simon interference (with unilateral stimuli) takes effect via the direct route of a dual-route model, Eriksen interference (even with arrow stimuli) takes effect mostly via the indirect route, and

the SR-mapping effect is a combination of slower processing via the indirect route and activation of the incorrect response via the direct route (c.f. Kornblum et al, 1990).

6.2.3.1 Conflict Adaptation or Congruency Sequence Effects

After defining the source of interference, there are limited means to assessing the domain-specificity of the control that resolves the resulting response conflict. A commonly used method is to measure conflict adaptation using 'congruency sequence effects' (CSEs, see Bream et al., 2014, for a recent review relevant to domain-specificity of control), in other words, how the congruency effect on the current trial is mediated by the type of interference on the previous trial. With interference by an irrelevant stimulus attribute, such as Eriksen or Simon interference, the interference effect is usually reduced on the following trial, which conflict-monitoring theory accounts for as an increase in cognitive control following detection of response conflict (Botvinick et al., 2001). The idea of using CSEs to assess the independence of control mechanisms is that, if control is domain-specific, then any increase in control relevant to resolving conflict on the previous trial will not reduce the subsequent interference effect on the current trial, assuming that the previous and current trial represent different sources of interference. As such, CSEs can be compared for consecutive trials with shared vs. independent sources of conflict (see Egner, 2008, for an account of experimental design criteria for such an analysis).

However, CSEs between trials are potentially problematic for assessing the independence of control mechanisms. Besides conflict-monitoring theory (Botvinick et al., 2001), there are alternative accounts for CSEs, some of which highlight a complication to using CSEs to assess the independence of control mechanisms. Crucially, CSEs can be accounted for by binding accounts (Mayr et al., 2003; Hommel, 2004; Hommel et al., 2004; Nieuwenhuis et al., 2006), meaning that responding will take longest when there is a partial overlap between stimulus and response features on subsequent trials, compared to complete repetitions or complete alternations, simply because bindings between stimulus and response features will be activated on one trial and need to be overcome during the next trial. Considering the standard (current-trial) congruency effects, this implies that the slowest responses are to *incongruent* trials that follow *congruent* trials, and the fastest to *congruent* trials that follow *congruent* trials – hence larger RT interference effects following congruent trials. Combining the conflict monitoring and binding theories into a learning

account, Verguts and Notebaert (2009) proposed that it is detection of response conflict (e.g. incongruent trials) that leads to a signal via ACC that strengthens all active associations (via Hebbian learning), reducing the effect of conflict on the following trial. However, the fundamental difference between these two accounts is that, like conflict-monitoring theory, Verguts and Notebaert (2009) assume that CSEs reflect conflict-driven control, rather than just the time taken to overcome inconvenient associations from the previous trial.

In fact, there is recent evidence using ERPs that the role of cognitive control in CSEs is limited to the earliest trials in an experiment. Von Gunten and coworkers found that throughout an Eriksen experiment, conflict related N2 enhancements following incongruent trials were gradually eliminated, despite consistent CSEs on RTs, and consistent current-trial congruency effects on N2 (von Gunten et al., 2018). This suggests that a large part of the assumed conflict adaptation effect must be accountable to binding alone, and that only the very first trials allow reliable assessment of between-trial control adjustments. Some studies have highlighted how between-trial conflict adaptation effects could be a by-product of within-trial control adjustments that correct response processing online (Burle et al., 2002; Ridderinkhof, 2002a), and some have even suggested that such within-trial control occurs simultaneously to the build-up of (rather than following detection of) response conflict (Scherbaum et al., 2011; Scherbaum et al., 2012).

In this thesis, the focus is *within*-trial control adjustment, using ERPs to compare conflict resolution with different sources of response conflict. This method poses its own problems because in both ERPs and RTs, conflict resolution with each type/source of interference partly reflects differences in the timing and magnitude of the associated response conflict (due to stimulus-complexity or dimensional overlap with the response). As such, the following discussion first illustrates differences in strategies for resolving different types of interference, and subsequently attempts a critical analysis of the extent to which the findings might reflect different magnitudes or temporal dynamics of the same control mechanisms in each case.

6.2.3.2 Domain-Specific Control Adjustments

The most efficient means to preventing any form of response conflict would be to close off the interfering path/associations completely, leaving only the relevant path active, but suppression/attenuation of interfering response activation is more feasible (e.g.

Ridderinkhof, 2002a). Dual-route models account for Simon interference as competition between response activation via direct (irrelevant locations) and indirect (relevant dimension) routes (e.g. de Jong, et al., 1994; Kornblum et al., 1990; Ridderinkhof et al., 1995), so the best means to reducing Simon interference would be by suppressing the direct route (e.g. Shaffer, 1965; Kornblum et al, 1990), yet the pattern of effects was not identical in chapters 2 and 3. With blocked interference (chapter 2), Simon effects were smaller than Eriksen effects, and there was no evidence of response conflict with Simon interference at contralateral N2. With mixed interference (chapter 3), such that Simon interference was unexpected, Simon interference was still smaller in RTs than Eriksen interference (with the compatible SR-mapping), there *was* evidence of response conflict at contralateral N2, and an enhanced midline N1 and N2 with all unilateral stimuli might be interpreted as online (reactive) suppression of the direct route. Possibly with blocked interference participants were able to suppress direct response activation proactively, which would explain the absence of response conflict at contralateral N2. Although these findings don't completely rule out the possibility that participants also suppressed the direct route with Eriksen interference (but to a lesser degree), they do at least suggest that there were differences in the strategies used to deal with each type of interference, whereby particularly Simon stimuli were associated direct route suppression.

Assuming that Eriksen interference takes effect predominantly via the indirect route (supported by findings in chapters 2 and 3), suppressing this entire route is far from optimal, as it would also slow selection of the correct response via the same route. Instead, it would be more effective if we could enhance attention to the target stimulus and attenuate attention to the flanker stimuli, both of which reflect conditional, direct route processing (supported by the findings in chapter 3). Support for control over relative attention to target and flankers in an Eriksen study comes from an experiment that used frequency-tagged EEG, in which target and flanker stimuli were tagged with different flicker frequencies (Scherbaum et al., 2011). Scherbaum and colleagues (2011) found that posterior EEG in incongruent trials demonstrated a within-trial power enhancement to the frequency associated with target compared to flanker stimuli, but only if the congruency or frequency tagging (and not both) switched from the previous trial. This finding has at least two important implications, both noted by Scherbaum et al. (2011, 2012). It suggests that cognitive control can influence attention to processing of specific stimulus attributes when

needed (within incongruent trials), and that within-trial changes in the strength of associations can carry-over to the following trial; but it also supports the idea that partial overlap between stimulus and response associations on consecutive trials form the most difficult sequences, as proposed by binding accounts (e.g. Hommel et al., 2004; Mayr et al., 2003), whereby flicker frequency unintentionally represents one of the stimulus attributes tied to such SR-bindings.

If a within-trial conflict-driven enhancement of attention to the target, and associated reduction in attention to the flankers, reflects increased control specific to conflict, then might we expect to find an enhanced fronto-central midline N2 with incongruent flankers? In chapters 2 and 3, midline N2 was not reliably enhanced with incongruent compared to congruent flankers, but in chapter 2 we did find evidence of a later component (N350) that was only present with incongruent flankers. N350 suggests that online control could be triggered by response conflict, but only at a very late stage. Scherbaum and colleagues (2011, 2012) note that their findings can be accounted for without the detection of response conflict, and their model accounts for this because goal representations bias processing toward relevant stimuli (top-down control), and these goals can be directly activated by stimulus attributes (bottom-up activation). As such, response conflict (reflected by lateral inhibition between active response representations) can still accumulate throughout the course of a trial, but does not need to be detected to increase goal activation.

However, there are at least two reasons why control adjustments (or updating of SR-bindings) might have happened earlier in the experiment by Scherbaum et al. (2011). The first is that their experiment did not include neutral trials, making it difficult to assess the extent to which contrast enhancements with incongruent trials were triggered by a pop-out effect, particularly because half of their congruent trials were an array of 5 identical stimuli, unlike for incongruent trials. In chapter 2, midline N2 was similar for neutral and incongruent flankers, and in chapter 3, midline N1 and N2 was significantly larger for neutral compared to incongruent flankers. This suggests that in all studies participants might have been using any available contrast between target and flanker stimuli as a cue to increase attention to target processing. The second reason why control processes might have been initiated earlier in the study by Scherbaum et al. (2011), is that participants were first made accustomed to the flicker frequencies associated with target/distractor locations during

presentation of a neutral cue that preceded the stimulus. Essentially, this gave participants the opportunity to enhance attention to the central target location (as well as the associated flicker frequency) prior to online response selection. Still, a comparison between the study by Scherbaum et al. (2011) and the Eriksen tasks in this thesis illustrates how control might respond to the earliest available information, and in other cases control might not be adjusted until response conflict has been detected.

6.2.3.3 Domain-Specific vs. Flexible Strategies

Considering the apparent differences in the determinants and temporal dynamics of control in all studies discussed so far, it might be the case that we employ specific strategies in dealing with each type of interference, whereby the nature of the interference depends on both the source of response conflict and the moment at which it can be detected/predicted. There are potential strategies that have been suggested by previous studies and tested in this thesis, but the domain-specificity of control depends upon the (limited) flexibility in the application of those strategies.

One strategy that can modulate interference effects is *voluntary control over the level of response activation* in preparation for the stimulus (Niemi & Nààtànen, 1981; see also Brown & Heathcote, 2005; Jahfari et al., 2012; Jennings & van der Molen, 2005; Hanes & Schall, 1996). A higher level of response activation can lead to faster RTs but increased errors, implying that the baseline level of activation in anticipation of the stimulus should not be dangerously close to the threshold for triggering a response. The LRP has provided some support for the hypothesis that participants control the level of response activation depending upon the speed-accuracy instruction (e.g. Band et al., 2003), and investigations into the neural mechanisms underlying the speed-accuracy trade-off (SAT) have also suggested that baseline response activation is adjusted, corresponding to an increase or decrease in the amount of decision processing needed before a response is triggered (see Bogacz et al., 2010, for a review). Some have proposed that participants temporarily suppress response activation when interference is expected (Band & van Boxtel, 1999; de Jong, 1995; Jennings & van der Molen, 2005; Klein et al., 2014; Stürmer et al., 2002), for which we found some support with an *incompatible* compared to a compatible SR-mapping in chapter 3, and with *mixed* compared to blocked SR-mappings in chapter 4. However, such

a mechanism should be considered as domain-general, as it does not depend on the source or type of conflict, and is exerted on the same brain areas (presumably the motor cortex).

But the level of response activation does not need to be equally distributed. Potentially, the baseline activation level could differ between hemispheres – for example if recent responding is biased toward one hand (for a review of factors that influence the level of response activation, see Niemi & Näätänen, 1981). Rather than a general suppression of response activation, others have suggested that we exert specific suppression of a primed response when expecting (de Jong, 1995; Klein et al., 2014) or following (Stürmer et al., 2002) response conflict. If suppression is specific to just one type of interference, then this might be interpreted as domain-specific control, such as suppressing the direct route (e.g. with Simon stimuli in chapter 3) or inhibiting processing of noise stimuli (e.g. with Eriksen stimuli in chapter 3). Alternatively, it might be the case that the reduced amplitude of N2 with Eriksen compared to Simon stimuli in chapter 3 reflected a general reduction to response activation, particularly as this difference was reliable in both contralateral and ipsilateral components. Such an interpretation subtracts from the conclusion that participants suppressed the direct route with Simon stimuli, but again emphasizes different control strategies with each type of interference.

In more complex tasks, besides a general reduction to response activation, other strategies are likely to benefit performance, such as influence over the relative activation of individual task schemas (see Monsell, 2003; Jennings & van der Molen, 2005). As noted by Egner (2008), task-switching designs are not well suited to comparing control between sources of conflict because it is difficult to dissociate between levels of interference. For example, task-switching costs are often accounted for by mutual inhibition at the task level (c.f. Norman & Shallice, 1986), such that activating one task will be much more difficult if the other task has just been activate. The task-switching literature can account for the findings in chapter 4, whereby switching from an incompatible to a compatible task demonstrated the most conflict, because switch costs are usually larger when switching to a stronger task (e.g. Allport et al., 1994; Yeung & Monsell, 2003). Also, N-120 appears to be specifically enhanced with switches to the compatible mapping, in line with the idea that control processes are required to overcome inhibition of the stronger task (Allport et al., 1994). Specifically, with mixed SR-mappings in chapter 4, preparation was biased toward the incompatible mapping, in line with the idea that preparatory control over task goals can bias responding (e.g.

Jennings et al., 2002; Jennings & van der Molen, 2005; Verbruggen, 2016). Intuitively, the mixed SR-mapping task in chapter 4 could also be interpreted as a single task, because each stimulus (gaze x eccentricity) was associated with one response. Nevertheless, the robust effects of mixed compared to blocked SR-mappings and the bias to the weaker SR-mapping (chapter 4) suggest that both conflict and its resolution reflected competition between tasks. Although the studies presented in chapters 4 and 5 are not well suited to testing the domain-specificity of control, they do highlight how interference reflects competition between task goals, which will be much higher with mixed task rules.

Assuming that control influences the relative activation of task goals (1 or more), even if it takes effect at a different moment and following different triggers, then perhaps the control mechanisms themselves are still not independent. For example, if an enhancement to cognitive control implies a general strengthening of all task relevant associations (e.g. Verguts & Notebaert, 2009; Scherbaum et al., 2012), then the mechanism itself is essentially the same, but might have influence on different sources of conflict. Sources of conflict can be defined not only along the lines of dual-route models (e.g. Kornblum et al., 1990), but as multiple potential sources of competing response activation (e.g. Ridderinkhof 2014), or even as specific representations or associations. Furthermore, depending on the dynamics of connections in the brain, a strengthening of task goals could imply both activation of relevant representations and inhibition of irrelevant representations. Theoretically, the temporal properties of such a signal could also be different - for example, continuous, proactive, reactive to stimulus attributes, reactive to response conflict, within-trials, or between-trials (see also Scherbaum et al., 2012, for suggestions regarding the timing of control adjustments) – without implying that each control adjustment reflected a different mechanism. However, while such an account could present cognitive control as domain-general, it also emphasizes tremendous flexibility of control adjustments in terms of both its timing and its determinants.

6.2.3.4 Interim Conclusion 3: The Independence of Control Strategies

A critical analysis of the findings presented in this thesis, in the context of other studies assessing the domain-specificity of control, highlights the theoretical nature of this final research question. Regardless of whether the trigger to increase control is response conflict (e.g. Botvinick et al., 2011) or interactions between stimulus and response bindings (e.g.

Scherbaum et al., 2012), if all control adjustments reflect a general strengthening of task goals, then such goals might incorporate the specific strategies assumed to resolve each source of conflict. For example, the strategies noted that might be interpreted as domain-specific are: a general suppression of response activation (e.g. with increasing task complexity); and a specific suppression of direct response activation (e.g. with task-irrelevant direct response priming). However, if we assume that control always takes effect at the task level, theoretically a general strengthening of task goals might incorporate such general or specific suppression. Further research could attempt to elucidate the dependence vs. independence of these potentially separate mechanisms of control.

6.3 Conclusions, Limitations, and Future Directions

This thesis attempted to answer three major questions on the roles of stimulus-response compatibility and cognitive control in decision-making, using performance and ERP measures of interference and control. While it is clear that none of these questions can be given a definitive answer, we did gain insight into all three. We managed to dissociate ERP measures of conflict and reactive online control, and found relatively late stimulus-locked or response-locked midline components associated with high conflict conditions that were paired with corrections to response preparation reflected over motor cortex. We also found support for top-down preparatory control over task goals (chapters 4 and 5) that revealed the dynamic interplay between proactive and reactive control, whereby over-preparation for the most probable (chapter 5) or most difficult (chapter 4) task resulted in reactive control updates in unexpected conditions. Finally, we found support that Simon and Eriksen interference reflect different sources of response conflict, and potentially different mechanisms of control, depending on the task set. With blocked interference, it seems that to a large extent response conflict could be attenuated by preparatory control, particularly Simon interference, presumably reflecting suppression of the direct route. With mixed interference, Eriksen and Simon interference were less predictable, and we saw no reliable evidence of reactive control over Eriksen interference. As such, Eriksen interference is most likely resolved by proactive strategies, such as increased focus on target compared to flanker stimuli, whereas Simon interference is likely resolved by a later, reactive suppression of the direct route as soon as participants can predict possible interference by location. Finally, although the results justify the distinction and interplay between proactive and reactive

control, including their respective triggers (global vs. stimulus attributes or response conflict), the mechanisms resulting from proactive and reactive control adjustments might be essentially the same: strengthen task-goals. While confirmation of this idea is beyond the scope of this thesis, research using computational modeling, with high-density EEG or MEG offers more potential.

Additional insight into the research questions could be gained by combining behavioral and ERP measures with other approaches to these problems, such as computational modeling to predict interference effects via multiple routes, with or without the explicit inclusion of conflict monitoring (e.g. Scherbaum et al., 2012), but then with different sources of interference. Potentially, reactive control is flexible enough to use either salient stimulus attributes or detection of response conflict, depending on the complexity of the task and the strategy selected, and future simulation studies might offer insight into which strategies are the most likely used and the most efficient. Also, refined statistical analyses that include data from all trials (such as mixed effects models), coding trial number, current, previous, and block congruency, could help to elucidate separate influences of practice, expectation, and within- and between-trial control.

When the temporal dynamics of conflict resolution are difficult to define and it is unclear which ERP components are most likely to reflect cognitive control processes, it could be more fruitful to broaden the temporal window and to assess spectral EEG instead of ERPs. Cognitive control has been associated not only with enhancements to midline frontal components, but also to midline frontal theta power (4-8 Hz, see Cavanagh & Frank, 2014, for a review). The dominant frequency in stimulus-locked ERPs is normally in the alpha range (8-12 Hz, see Makeig et al., 2002 for an example of the spectral properties of ERPs), but with a high cognitive workload, alpha power has been shown to decrease and theta power to increase (see Borghini et al., 2014, for a review), which could manifest as a wider N2 in stimulus-locked ERPs. Future studies assessing both ERPs and spectral EEG comparing congruent, neutral, and incongruent Eriksen trials could help to dissociate the effects of stimulus processing, conflict and control on response slowing.

Importantly, increases in frontal theta power have been associated with response slowing (e.g. Scherbaum & Dshemuchadse. 2013), and increases in alpha power over contralateral visual processing areas have been associated with inhibition of lateralized incongruent flankers (Janssens et al., 2018), which is in line with the idea that increased

attentional control within specific brain networks could be measured by the ratio between theta and alpha power (see also Clayton et al., 2015, for a review of oscillations related to attention). However, there are still many unanswered questions regarding the origins of EEG signals (c.f. Cohen, 2017), and much more research is needed into the relationship between ERPs and spectral EEG (but see Makeig et al, 2002; Yeung, Bogacz, et al., 2004), especially in relation to modulation of attention and control. For example, could we use a combination of ERPs and spectral EEG to differentiate between bottom-up (e.g. stimulus-driven, c.f. binding accounts) and top-down (e.g., control, increased attention, selective inhibition) processing?

As concluded by Mansouri et al. (2009), conflict detection via ACC does not appear to consistently predict behavioral adjustments. In order to assess whether conflict monitoring is unnecessary to control adjustments (c.f. Scherbaum et al., 2012), researchers could make use of MEG, or ERPs recorded simultaneously with fMRI. MFC and ACC have commonly been associated with performance monitoring (e.g. Botvinick et al., 2007; Ridderinkhof et al., 2004), but others have suggested that ACC might instead reflect regulation of control (e.g. Roelofs et al., 2006) or be involved in response selection (Mansouri et al., 2009). Complex tasks such as unpredictable mixed SR-mappings in combination with MEG or combined fMRI and ERPs could help to differentiate between the three dissociable control mechanisms identified in chapter 4 (reflected by N2, N-120, and CRN). Braver, (2009, 2012) refers to some of the potential factors influencing individual differences in proactive and reactive control capacity, and also in the preference for one type of control over the other. Proactive control is assumed to be effortful and to rely upon motivation (e.g. Braver, 2012; Jennings & van der Molen, 2005), but the mechanisms of proactive control are still relatively unknown. Potentially, both inter-individual and intra-individual variation in proactive control also reflect a combination of functional connectivity between brain areas and available resources, which may or may not be limited (for a review, see Inzlicht, et al., 2014). Thus one unanswered question is whether we could reduce the effort required by both reactive and proactive control by use of cognitive training to increase neural efficiency. As a consequence, would we see a reduction to reactive control, to proactive control, to both, or is it possible to train each of these mechanisms independently?

To sum, cognitive control is immensely flexible, relying upon general and specific strategies. Despite some clear benefits to specific strategies in certain conditions, control could theoretically utilize the same strategy for multiple sources of conflict: strengthen task

goals in order to activate relevant associations and inhibit competing representations. The most likely means by which a single mechanism of control might achieve the flexibility implied by the findings in this thesis and other studies discussed is by making continuous adjustments to control based on the available input. As such, control could better be defined in terms of learning accounts (e.g. Verguts & Notebaert, 2009), but then with constant updating, as suggested by Scherbaum et al. (2012). The role of conflict might play a smaller role than the general consensus suggests, particularly in situations that are more complex than the typical conflict tasks. My intuition is that control is primarily proactive, and how we regulate control reactively will depend upon motivation, functional connectivity, cognitive resources, and how we have set up and maintained task goals.

References

Allport, D. A., Styles, E. A., & Hsieh, S. (1994). Shifting intentional set: Exploring the dynamic control of tasks. In C. Umiltà, & M. Moscovitch (Eds.), *Attention and Performance XV: Conscious and non-conscious information processing* (pp. 421-452). Cambridge, MA: MIT Press.

Ansorge, U. (2003). Spatial Simon effects and compatibility effects induced by observed gaze direction. *Visual Cognition*, *10*, 363-383.

Band, G. P. H., & Van Boxtel, G. J. M. (1999). Inhibitory motor control in stop paradigms: review and reinterpretation of neural mechanisms. *Acta psychologica*, *101*(2), 179-211.

Band, G.P., Ridderinkhof, K.R., & van der Molen, M.W. (2003). Speed-accuracy modulation in case of conflict: The roles of activation and inhibition. *Psychological Research, 67*, 266-279.

Bartholow, B. D., Pearson, M. A., Dickter, C. L., Sher, K. J., Fabiani, M., & Gratton, G. (2005). Strategic control and medial frontal negativity: Beyond errors and response conflict. *Psychophysiology*, *42*(1), 33-42.

Berkman, E. T., Kahn, L. E., & Merchant, J. S. (2014). Training-induced changes in inhibitory control network activity. The Journal of Neuroscience, 34, 149-157.

Blair, R. C., & Karniski, W. (1993). An alternative method for significance testing of waveform difference potentials. *Psychophysiology, 30*, 518-524.

Bogacz, R., Wagenmakers, E. J., Forstmann, B. U., & Nieuwenhuis, S. (2010). The neural basis of the speed–accuracy tradeoff. *Trends in neurosciences*, *33*(1), 10-16.

Borghini, G., Astolfi, L., Vecchiato, G., Mattia, D., & Babiloni, F. (2014). Measuring neurophysiological signals in aircraft pilots and car drivers for the assessment of mental workload, fatigue and drowsiness. *Neuroscience & Biobehavioral Reviews, 44*, 58-75.

Botvinick, M. M., Braver, T. S., Barch, D. M., Carter, C. S., & Cohen, J. D. (2001). Conflict monitoring and cognitive control. *Psychological review*, *108*(3), 624.

Botvinick, M. M., Cohen, J. D., & Carter, C. S. (2004). Conflict monitoring and anterior cingulate cortex: an update. *Trends in cognitive sciences, 8*(12), 539-546.

Botvinick, M., Nystrom, L. E., Fissell, K., Carter, C. S., & Cohen, J. D. (1999). Conflict monitoring versus selection for-action in anterior cingulate cortex. Nature, 402, 179–181.

Braem, S., Abrahamse, E. L., Duthoo, W., & Notebaert, W. (2014). What determines the specificity of conflict adaptation? A review, critical analysis, and proposed synthesis. *Frontiers in psychology, 5*, 1134.

Braver, T. S. (2012). The variable nature of cognitive control: a dual mechanisms framework. *Trends in cognitive sciences*, *16*(2), 106-113.

Braver, T. S., Gray, J. R., & Burgess, G. C. (2007). Explaining the many varieties of working memory variation: Dual mechanisms of cognitive control. In A. R. A. Conway, C. Jarrold, M. J. Kane, A. Miyake, & J. N. Towse (Eds.), *Variation in working memory* (pp. 76-106). Oxford: Oxford University Press.

Braver, T. S., Paxton, J. L., Locke, H. S. & Barch, D. M. (2009). Flexible neural mechanisms of cognitive control with human prefrontal cortex. Proc Natl Acad Sci U.S.A., 106, 7351-7356.

Brown, S., & Heathcote, A. (2005). A ballistic model of choice response time. *Psychological Review*, *112*(1), 117.

Burle, B., Allain, S., Vidal, F., & Hasbroucq, T. (2005). Sequential compatibility effects and cognitive control: Does conflict really matter? *Journal of Experimental Psychology: Human Perception and Performance, 31*, 831-837.

Burle, B., Possamaï, C. A., Vidal, F., Bonnet, M., & Hasbroucq, T. (2002). Executive control in the Simon effect: an electromyographic and distributional analysis. *Psychological research, 66*(4), 324-336.

Burle, B., Vidal, F., Tandonnet, C., & Hasbroucq, T. (2004). Physiological evidence for response inhibition in choice reaction time tasks. *Brain and Cognition, 56*, 153-164.

Carbonnell, L., Hasbroucq, T., Grapperon, J., & vidal, F. (2004). Response selection and motor areas: e behavioral and electrophysiological study. *Clinical Neurophysiology, 115*, 2164-2174.

Carbonnell, L., Ramdani, C., Meckler, C., Burle, B., Hasbroucq, T., & Vidal, F. (2013). The N-40: An electrophysiological marker of response selection. *Biological Psychology, 93*, 231-236.

Carriero, L., Zalla, T., Budai, R., & Battaglini, P. P. (2007). Inhibition of wrong responses and conflict resolution: an electroencephalogram study. *NeuroReport, 18*, 793-796.

Carter, C. S., & Van Veen, V. (2007). Anterior cingulate cortex and conflict detection: an update of theory and data. *Cognitive, Affective, & Behavioral Neuroscience, 7*(4), 367-379.

Carter, C. S., Braver, T. S., Barch, D. M., Botvinick, M. M., Noll, D., & Cohen, J. D. (1998). Anterior cingulate cortex, error detection, and the online monitoring of performance. *Science*, *280*(5364), 747-749.

Carter, C. S., Macdonald A.M., Botvinick, M., Ross, L. L., Stenger, V. A., Noll, D., et al. (2000). Parsing executive processes: Strategic vs. evaluative functions of the anterior cingulate cortex. *Proceedings of the Nation Academy of Sciences, U.S.A., 97*, 1944-1948.

Cavanagh, J. F., & Frank, M. J. (2014). Frontal theta as a mechanism for cognitive control*. Trends in Cognitive Sciences, 18*(8), 414– 421.

Cespón, J., Galdo-Álvarez, S., & Díaz, F. (2012). The Simon effect modulates N2cc and LRP but not the N2pc component. *International Journal of Psychophysiology, 84(2)*, 120-129.

Cespón, J., Galdo-Álvarez, S., & Díaz, F. (2013). Similarities and differences between interference from stimulus position and from direction of an arrow: Behavioral and event-related potential measures. *International Journal of Psychophysiology, 90(2)*, 180-189.

Christensen, C. A., Ivkovich, D., & Drake, K. J. (2001). Late positive ERP peaks observed in stimulus-response compatibility tasks tested under speed-accuracy instructions. *Psychophysiology, 38*, 404-416.

Cohen-Kdoshay, O., & Meiran, N. (2009). The representation of instructions operates like a prepared reflex. *Experimental Psychology*, *56*, 128-133.

Cohen, M. X. (2017). Where does EEG come from and what does it mean?. *Trends in neurosciences, 40*(4), 208-218.

Cole, M. W., Bagic, A., Kass, R., & Schneider, W. (2010). Prefrontal dynamics underlying rapid instructed task learning reverse with practice. *Journal of Neuroscience, 30*(42), 14245-14254.

Coles, M. G., Gratton, G., Bashore, T. R., Eriksen, C. W., & Donchin, E. (1985). A psychophysiological investigation of the continuous flow model of human information processing. Journal of Experimental Psychology: *Human Perception and Performance, 11*(5), 529.

Coles, M.G.H. (1989). Modern mind-brain reading: psychophysiology, physiology, and cognition. Psychophysiology, 26(3), 251-269.

Crowley, K. E., & Colrain, I. M. (2004). A review of the evidence for P2 being an independent component process: age, sleep and modality. *Clinical neurophysiology*, *115*(4), 732-744.

de Jong, R. (1995). Strategical determinants of compatibility effects with task uncertainty. *Acta Psychologica*, *88*(3), 187-207.

de Jong, R., Liang, C. C., & Lauber, E. (1994). Conditional and unconditional automaticity: a dual-process model of effects of spatial stimulus-response correspondence. *Journal of Experimental Psychology: Human Perception and Performance, 20(4)*, 731.

de Jong, R., Wierda, M., Mulder, G., & Mulder, L. G. M. (1988). Use of partial stimulus information in response processing. *Journal of Experimental Psychology: Human Perception and Performance, 14*, 682-692.

De Pisapia, N., & Braver, T. S. (2006). A model of dual control mechanisms through anterior cingulate and prefrontal cortex interactions. *Neurocomputing*, *69*(10), 1322-1326.

Delorme, A., & Makeig, S. (2004). EEGLAB: an open source toolbox for analysis of single-trial EEG dynamics. *Journal of Neuroscience Methods, 134*, 9-21.

Donchin, E., & Coles, M. G. H. (1988). Is the P300 component a manifestation of context updating? The Behavioral and Brain Sciences, 11, 357-374.

Donkers, F. C. L., & van Boxtel, G. J. M. (2004). N2 in go/no-go tasks reflects conflict monitoring not response inhibition. *Brain and Cognition, 56*, 165-176.

Dumontheil, I., Thompson, R., & Duncan, J. (2011). Assembly and use of new task rules in fronto-parietal cortex. *Journal of Cognitive Neuroscience, 23*(1), 168-182.

Duncan, J. (1978). Response selection in spatial choice reaction: Further evidence against associative models. *Quarterly Journal of Experimental Psychology, 30*, 429-440.

Egner, T. (2008). Multiple conflict-driven control mechanisms in the human brain. *Trends in cognitive sciences, 12*(10), 374-380.

Eimer, M. (1995). Stimulus-response compatibility and automatic response activation: Evidence from psychophysiological studies. *Journal of Experimental Psychology: Human Perception and Performance, 21*, 837–854.

Eimer, M. (1996). The N2pc component as an indicator of attentional selectivity. *Electroencephalography and clinical neurophysiology*, *99*(3), 225-234.

Eimer, M. (2014). The neural basis of attentional control in visual search. *Trends in cognitive sciences*, *18*(10), 526-535.

Eimer, M., & Schlaghecken, F. (1998). Effects of masked stimuli on motor activation: behavioral and electrophysiological evidence. *Journal of Experimental Psychology: Human Perception and Performance, 24(6)*, 1737.

Eimer, M., & Schlaghecken, F. (2003). Response facilitation and inhibition in subliminal priming. *Biological psychology*, *64*(1), 7-26.

Eimer, M., Hommel, B., Prinz, W. (1995). S-R compatibility and response selection. *Acta Psychologica, 90*, 301-313.

Eriksen, B. A., & Eriksen, C. W. (1974). Effects of noise letters upon the identification of a target letter in a nonsearch task. *Perception and Psychophysics, 16,* 143-149.

Eriksen, C. W., & Schultz, D. W. (1979). Information processing in visual search: A continuous flow conception and experimental results. *Perception & Psychophysics, 25*(4), 249-263.

Eriksen, C. W., Coles, M. G., Morris, L. R., & O'hara, W. P. (1985). An electromyographic examination of response competition. *Bulletin of the Psychonomic Society*, *23*(3), 165-168.

Exner, S. (1879). Physiologie der Grosshirnrinde. In L. Hermann (Ed.), *Handbuch der Physiologie, vol. 2, part 2*, pp. 189–350. Leipzig: Vogel.

Falkenstein, M. (2002). ERP correlates of erroneous performance. In M. Ullsperger & M. Falkenstein (Eds.), *Errors, Conflicts, and the Brain: Current Opinions on Performance Monitoring* (pp. 5-14). Leipzig: Max Planck Institute of Cognitive Neuroscience, ISBN 3-936816-16-6.

Falkenstein, M., Hohnsbein, J., Hoorman, J., & Blanke, L. (1991). Effects of crossmodal divided attention on late ERP components: II. Error processing in choice reaction tasks. Electroencephalography and Clinical Neurophysiology, 78, 447–455.

Falkenstein, M., Hoormann, J., & Hohnsbein, J. (1999). ERP components in Go/Nogo tasks and their relation to inhibition. *Acta Psychologica, 101*, 267-291.

Falkenstein, M., Hoormann, J., & Hohnsbein, J. (2002). Inhibition-related ERP components: variation with modality, age and time-on-task. *Journal of Psychophysiology, 16*, 167-175.

Fitts P. M. (1958). Engineering psychology. *Annual Review of Psychology*, *9*, 267–94.

Fitts, P. M., & Deininger, R. L. (1954). SR compatibility: correspondence among paired elements within stimulus and response codes. *Journal of experimental psychology*, *48*(6), 483.

Fitts, P. M., & Seeger, C. M. (1953). SR compatibility: spatial characteristics of stimulus and response codes. *Journal of experimental psychology, 46*(3), 199.

Folstein, J. R., & van Petten, C. (2008). Influence of cognitive control and mismatch on the N2 component of the ERP: A review. *Psychophysiology, 45*, 152-170.

Forstmann, B. U., Ridderinkhof, K. R., Kaiser, J., & Bledowski, C. (2007). At your own peril: An ERP study of voluntary task set selection processes in the medial frontal cortex. *Cognitive, Affective, & Behavioral Neuroscience, 7 (4)*, 286-296.

Gajewski, P. D., Kleinsorg, T., & Falkenstein, M. (2010). Electrophysiological correlates of residual switch costs. Cortex, 46, 1138-1148.

Gajewski, P. D., Stoerig, P., & Falkenstein, M. (2008). ERP—correlates of response selection in a response conflict paradigm. *Brain research, 1189*, 127-134.

Gehring, W. J., Goss, B., Coles, M. G. H., Meyer, D. E., & Donchin, E. (1993). A neural system for error detection and compensation. *Psychological Science, 4*, 385–390.

Gilbert, S.J. and Shallice, T. (2002) Task switching: a PDP model. *Cognitive Psychology, 44*, 297–337.

Gratton, G., Coles, M. G., & Donchin, E. (1992). Optimizing the use of information: strategic control of activation of responses. *Journal of Experimental Psychology: General*, *121*(4), 480-506.

Gratton, G., Coles, M. G., Sirevaag, E. J., Eriksen, C. W., & Donchin, E. (1988). Pre-and poststimulus activation of response channels: a psychophysiological analysis. *Journal of Experimental Psychology: Human perception and performance*, *14*(3), 331-344.

Guthrie, D., & Buchwald, J. S. (1991). Significance testing of difference potentials. *Psychophysiology, 28*, 240-244.

Hanes, D. P., & Schall, J. D. (1996). Neural control of voluntary movement initiation. *Science, 274*, 427-430.

Hansen, J. C., & Hillyard, S. A. (1980). Endogeneous brain potentials associated with selective auditory attention. *Electroencephalography and clinical neurophysiology*, *49*(3), 277-290.

Hasbroucq, T., & Guiard, Y. (1991). Stimulus-response compatibility and the Simon effect: Toward a conceptual clarification. *Journal of Experimental Psychology: Human Perception and Performance, 17*, 246-266.

Hedge, A., & Marsh, N. W. A. (1975). The effect of irrelevant spatial correspondence on two-choice response-time. *Acta Psychologica, 39*, 427-439.

Heil, M., Osman, A., Wiegelmann, J., Rolke, B., & Hennighausen, E. (2000). N200 in the Eriksen-Task: Inhibitory executive processes? *Journal of Psychophysiology, 14*, 218-225.

Heister, G., & Schroeder-Heister, P. (1994). Spatial S-R compatibility: Positional instructions vs. compatibility instruction. *Acta Psychologica, 85*, 15-24.

Hohnsbein, J., Falkenstein, M., Hoormann, J., & Blanke, L. (1991). Effects of cross-modal divided attention on late ERP components. II. Error processing in choice reaction tasks. *Electroencephalography and clinical Neurophysiology, 78*, 447-455.

Homan, R. W., Herman, J., & Purdy, P. (1987). Cerebral location of international 10-20 system electrode placement. *Electroencephalography and Clinical Neurophysiology, 66*, 376-382.

Hommel, B. (1993). The relationship between stimulus processing and response selection in the Simon task: Evidence for a temporal overlap. *Psychological Research, 55*, 280-290.

Hommel, B. (1995). Stimulus-response compatibility and the Simon effect: Toward an empirical clarification. *Journal of Experimental Psychology: Human Perception and Performance, 21*, 764-755.

Hommel, B. (2000). The prepared reflex: Automaticity and control in stimulus-response translation. In S. Monsell & J. Driver (Eds.), *Control of cognitive processes: Attention and performance*, MIT Press, Cambrigde, pp.247-274.

Hommel, B. (2004). Event files: Feature binding in and across perception and action. *Trends in cognitive sciences*, *8*(11), 494-500.

Hommel, B., Proctor, R. W., & Vu, K-P. L (2004). A feature integration account of sequential effects in the Simon task. *Psychological Research, 68*, 1-17.

Ikeda, A., Lüders, H. O., Shibasaki, H. Collura, T. F., Burgess, R. C., Morris, H. H., & Hamano, T. (1995). Movement-related potentials associated with bilateral simultaneous and unilateral movements recorded from human supplementary motor area. *Electroencephalography and Clinical Neurophysiology, 95*, 323–334.

Inzlicht, M., Schmeichel, B. J., & Macrae, C. N. (2014). Why self-control seems (but may not be) limited. *Trends in cognitive sciences, 18(3),* 127-133.

Jahfari, S., Verbruggen, F., Frank, M. J., Waldorp, L. J., Colzato, L., Ridderinkhof, K. R., & Forstmann, B. U. (2012). How preparation changes the need for top-down control of the basal ganglia when inhibiting premature actions. The Journal of Neuroscience, 32, 10870-10878.

James, W. (1890). *Principles of Psychology*. New York: Holt.

Janssens, C., De Loof, E., Boehler, C. N., Pourtois, G., & Verguts, T. (2018). Occipital alpha power reveals fast attentional inhibition of incongruent distractors. *Psychophysiology, 55*(3), e13011.

Jennings, J. R., & van der Molen, M. W. (2005). Preparation for speeded action as a psychophysiological concept. *Psychological bulletin*, *131*(3), 434.

Jennings, J. R., Van Der Molen, M. W., Van der Veen, F. M., & Debski, K. B. (2002). Influence of preparatory schema on the speed of responses to spatially compatible and incompatible stimuli. *Psychophysiology, 39*(4), 496-504.

Jentzsch, I., & Leuthold, H. (2005). Response conflict determines sequential effects in serial response time tasks with short response-stimulus intervals. *Journal of Experimental Psychology: Human Perception and Performance, 31*, 731-748.

Kahneman, D., & Treisman, A. (1984). Changing views of attention and automaticity. in r. parasuraman & dr davies (eds.), Varieties of attention (pp. 29-61).

Kahneman, D., Treisman, A., & Gibbs, B. J. (1992). The reviewing of object files: Object-specific integration of information. *Cognitive psychology*, *24*(2), 175-219.

Klein, P. A., Petitjean, C., Olivier, E., & Duque, J. (2014). Top-down suppression of incompatible motor activations during response selection under conflict. *NeuroImage*, *86*, 138-149.

Kopp, B., Mattler, U., Goertz, & Rist, F. (1996). N2, P3 and the lateralized readiness potential in a nogo task involving selective response priming. *Electroencephalography & Clinical Neurophysiology, 99*, 19-27.

Kopp, B., Rist, F., & Mattler, U. (1996). N200 in the flanker task as a neurobehavioral tool for investigating executive control. *Psychophysiology, 33*, 282-294.

Kornblum, S., Hasbroucq, T., & Osman, A. (1990). Dimensional overlap: cognitive basis for stimulus-response compatibility--a model and taxonomy. *Psychological review*, *97*(2), 253.

Kornhuber, H. H., & Deecke, L. (1965). Hirnpotentialänderungen bei Willkürbewegungen und passiven Bewegungen des Menschen: Bereitschaftspotential und reafferente Potentiale. *Pflüger's Archiv für die gesamte Physiologie des Menschen und der Tiere, 284*(1), 1-17.

Kutas, M., McCarthy, G., & Donchin, E. (1977). Augmenting mental chronometry: The P300 as a measure of stimulus evaluation time. Science, 197, 792-795.

Lamers, M. J., & Roelofs, A. (2011). Attentional control adjustments in Eriksen and Stroop task performance can be independent of response conflict. *Quarterly Journal of Experimental Psychology, 64*(6), 1056-1081.

Leuthold, H. (2011). The Simon effect in cognitive electrophysiology: a short review. *Acta psychologica*, *136*(2), 203-211.

Leuthold, H. & Jentzsch, I. (2002). Distinguishing neural sources of movement preparation and execution: An electrophysiological analysis. Biological Psychology, 60, 173-198.

Leuthold, H., & Schröter, H. (2006). Electrophysiological evidence for response priming and conflict regulation in the auditory Simon task. *Brain Research*, *1097*(1), 167-180.

Leuthold, H., & Sommer, W. (1998). P300 latency and postperceptual processes. Psychophysiology, 35, 34-46.

Lindsen, J. P., & De Jong, R. (2010). Distinguishing between the partial-mapping preparation hypothesis and the failure-to-engage hypothesis of residual switch costs. *Journal of Experimental Psychology: Human Perception and Performance, 36.*1207-1226.

Logan, G. D. (1985). Executive control of thought and action. *Acta Psychologica*, *60*(2-3), 193-210.

Logan, G. D., & Cowan, W. B. (1984). On the ability to inhibit thought and action: A theory of an act of control. *Psychological Review, 91*, 295-327.

Lu, C. H., & Proctor, R. W. (1995). The influence of irrelevant location information on performance: A review of the Simon and spatial Stroop effects. *Psychonomic bulletin & review*, *2*(2), 174-207.

Lu, C.H., & Proctor, R. W. (1994). Processing of an irrelevant location dimension as a function of the relevant stimulus dimension. *Journal of Experimental Psychology: Human Perception and Performance, 20*, 286-298.

Luck, S. J., & Hillyard, S. A. (1994). Electrophysiological correlates of feature analysis during visual search. *Psychophysiology*, *31*(3), 291-308.

Luck, S.J., Girelli, M., McDermott, M.T., & Ford, M.A. (1997). Bridging the gap between monkey neurophysiology and human perception: An ambiguity resolution theory of visual selective attention. *Cognitive Psychology, 33*, 64-87.

Lungu, O. V., Liu, T., Waechter, T., Willingham, D., T., & Ashe, J. (2007). Strategic modulation of cognitive control. Journal of Cognitive Neuroscience, 19, 1302-1315.

MacDonald, A. W., Cohen, J. D., Stenger, V. A., & Carter, C. S. (2000). Dissociating the role of the dorsolateral prefrontal and anterior cingulate cortex in cognitive control. *Science, 288*(5472), 1835-1838.

Makeig, S., Westerfield, M., Jung, T.P., Enghoff, S., Townsend, J., Courchesne, E., & Sejnowski, T.J. (2002) Dynamic brain sources of visual evoked responses. *Science, 295*:690 – 694.

Mansfield, K. L., van der Molen, M. W., & van Boxtel, G. J. M. (2012). Proactive and reactive control in S-R compatibility: A brain potential analysis. *Psychophysiology, 49*, 756-769.

Mansfield, K. L., van der Molen, M. W., Falkenstein, M., & van Boxtel, G. J. (2013). Temporal dynamics of interference in Simon and Eriksen tasks considered within the context of a dual-process model. *Brain and cognition*, *82*(3), 353-363.

Mansouri, F. A., Tanaka, K., & Buckley, M. J. (2009). Conflict-induced behavioural adjustment: a clue to the executive functions of the prefrontal cortex. *Nature Reviews Neuroscience, 10*(2), 141.

Masaki, H., Falkenstein, M., Sturmer, B., Pinkpank, T., & Sommer, W. (2007). Does the error negativity reflect response conflict strength? Evidence from a Simon task. *Psychophysiology, 44*, 579-585.

Masaki, H., Takasawa, N., & Yamazaki, K. (2000). An electrophysiological study of the locus of the interference effect in a stimulus-response compatibility paradigm. *Psychophysiology, 37*, 464-472.

Mathewson, K. E., Beck, D. M., Ro, T., Maclin, E. L., Low, K. A., Fabiani, M., & Gratton, G. (2014). Dynamics of alpha control: preparatory suppression of posterior alpha oscillations by frontal modulators revealed with combined EEG and event-related optical signal. *Journal of cognitive neuroscience, 26*(10), 2400-2415.

Mayr, U., Awh, E., & Laurey, P. (2003). Conflict adaptation effects in the absence of executive control. *Nature neuroscience, 6*(5), 450.

McCarthy, G., & Donchin, E. (1981). A metric for thought: A comparison of P300 latency and reaction time. *Science, 211*, 77-80.

Meckler, C., Allain, S., Carbonnell, L., Hasbroucq, T., Burle, B., Vidal, F. (2010). Motor inhibition and response expectancy: A Laplacian ERP study. *Biological Psychology, 85*, 386-392.

Melara, R. D., Wang, H., Vu, K. –P., & Proctor, R. W. (2008). Attentional origins of the Simon effect: Behavioral and electrophysiological evidence. *Brain Research, 1215*, 147-159.

Monsell, S. (2003). Task switching. *Trends in cognitive sciences*, *7*(3), 134-140.

Näätänen, R., Gaillard, A. W., & Mäntysalo, S. (1978). Early selective-attention effect on evoked potential reinterpreted. *Acta psychologica*, *42*(4), 313-329.

Näätänen, R., Paavilainen, P., Rinne, T., & Alho, K. (2007). The mismatch negativity (MMN) in basic research of central auditory processing: a review. *Clinical neurophysiology, 118*(12), 2544-2590.

Neshige, R., Lüders, H., Shibasaki, H. (1988). Recording of movement related potentials in scalp and cortex in man. Brain, 111, 719-736.

Niemi, P., & Näätänen, R. (1981). Foreperiod and simple reaction time. *Psychological Bulletin*, *89*(1), 133.

Nieuwenhuis, S., Yeung, N., van den Wildenberg, W., & Ridderinkhof, K. R. (2003). Electrophysiological correlates of anterior cingulate function in a go/no-go task: Effects of

response conflict and trial type frequency. *Cognitive, Affective, & Behavioral Neuroscience, 3*, 17-26.

Norman, D. A., & Shallice, T. (1986). Attention to action. In R. J. Davidson, G. E. Schwartz, & D. Shapiro (Eds.), *Consciousness and self-regulation* (pp. 1–18). New York: Plenum Press.

Oostenveld, R., & Praamstra, P. (2001). The five percent electrode system for high-resolution EEG and ERP measurements. Clinical Neurophysiology, 112, 713-719.

Picard, N., & Strick, P. (1996). Motor areas of the medial wall: a review of their location and activation function. *Cerebral Cortex, 6*, 342-353.

Posner, M. I., & Snyder, C. R. (1975). Facilitation and inhibition in the processing of signals. In P. M. A. Rabbit & S. Dornic (Eds.), *Attention & Performance V* (pp. 669-682). New York: Academic Press.

Praamstra, P. (2007). Do's and don'ts with lateralized event related brain potentials. *Journal of Experimental Psychology: Human Perception and Performance, 33*, 497-502.

Praamstra, P., & Oostenveld, R. (2003). Attention and movement-related cortex activation: a high density EEG study of spatial stimulus-response compatibility. *Cognitive Brain Research, 16*, 309-322.

Praamstra, P., & Seiss, E. (2005). The neurophysiology of response competition: Motor cortex activation and inhibition following subliminal response priming. *Journal of Cognitive Neuroscience*, *17*(3), 483-493.

Praamstra, P., Kleine, B. U., & Schnitzler, A. (1999). Magnetic stimulation of the dorsal premotor cortex modulates the Simon effect. *NeuroReport, 10*, 3671-3674.

Proctor, R. W., & Vu, K. –P. L. (2002). Eliminating, magnifying, and reversing spatial compatibility effects with mixed location-relevant and irrelevant trials. In W. Prinz & B. Hommel (Eds.), *Common mechanisms in perception and action: Attention and Performance, Vol. XIX* (pp.443-473). Oxford UK: Oxford University Press.

Proctor, R. W., & Vu, K. P. L. (2010). Cumulative knowledge and progress in human factors. *Annual Review of Psychology*, *61*, 623-651.

Proctor, R., Vu, K. P. L., & Marble, J. G. (2003). Mixing location-relevant and irrelevant tasks: Spatial compatibility effects eliminated by stimuli that share the same spatial codes. *Visual Cognition*, *10*(1), 15-50.

Rabbitt, P. M. A., & Vyas, S. M. (1970). An elementary preliminary taxonomy for some errors in laboratory choice RT tasks. In A. F. Sanders (Ed.), *Attention & Performance III*. Amsterdam: North Holland

Ragot, R., & Renault, B. (1981). P300 as a function of S-R compatibility and motor programming. Biological Psychology, 13, 289-294.

Ridderinkhof, K. R. (2002a). Activation and suppression in conflict tasks: empirical clarification through distributional analyses. In W. Prinz & B. Hommel (Eds.), *Common Mechanisms in Perception and Action. Attention & Performance, Vol. XIX* (pp. 494–519). Oxford: Oxford University Press.

Ridderinkhof, K. R. (2002b). Micro- and macro-adjustments of task set: activation and suppression in conflict tasks. *Psychological Research, 66*, 312-323.

Ridderinkhof, K. R., & van der Molen, M. W. (1995). When global information and local information collide: a brain potential analysis of the locus of interference effects. *Biological Psychology, 41*, 29-53.

Ridderinkhof, K. R., Ullsperger, M., Crone, E. A., & Nieuwenhuis, S. (2004a). The role of the medial frontal cortex in cognitive control. *Science*, *306*(5695), 443-447.

Ridderinkhof, K. R., van den Wildenberg, W. P., Segalowitz, S. J., & Carter, C. S. (2004b). Neurocognitive mechanisms of cognitive control: the role of prefrontal cortex in action selection, response inhibition, performance monitoring, and reward-based learning. *Brain and cognition*, *56*(2), 129-140.

Ridderinkhof, K. R., van der Molen, M. W., & Bashore, T. R. (1995). Limits on the application of additive factors logic: Violations of stage robustness suggest a dual-process architecture to explain flanker effects on target processing. *Acta Psychologica*, *90*(1), 29-48.

Roelofs, A., Van Turennout, M., & Coles, M. G. (2006). Anterior cingulate cortex activity can be independent of response conflict in Stroop-like tasks. *Proceedings of the National Academy of Sciences, 103*(37), 13884-13889.

Rohrbaugh, J. W., & Gaillard, A. W. K. (1983). Sensory and motor aspects of the contingent negative variation. In A. W. K. Gaillard & W. Ritter (Eds.), *Tutorials in event-related potential research: The endogenous components* (pp. 269-310). Amsterdam: North-Holland.

Scherbaum, S., & Dshemuchadse, M. (2013) Higher response time increases theta energy, conflict increases response time. *Clin Neurophysiol 124*: 1477–1479.

Scherbaum, S., Dshemuchadse, M., Ruge, H., & Goschke, T. (2012). Dynamic goal states: adjusting cognitive control without conflict monitoring. *Neuroimage, 63*(1), 126-136.

Scherbaum, S., Fischer, R., Dshemuchadse, M., & Goschke, T. (2011). The dynamics of cognitive control: Evidence for within-trial conflict adaptation from frequency-tagged EEG. *Psychophysiology, 48*(5), 591-600.

Shaffer, L. H. (1965). Choice reaction with variable S-R mapping. *Journal of Experimental Psychology, 70*, 284–288.

Shiffrin, R.M., & Schneider, W. (1977). Controlled and automatic human information processing: II. Perceptual learning, automatic attending, and a general theory. *Psychological Review, 84*, 127-190.

Simon, J. R., & Rudell, A. P. (1967). Auditory S-R compatibility: Effect of an irrelevant cue on information processing. *Journal of Applied Psychology, 51*, 300-304.

Smid, H. G. O. M., Mulder, G., & Mulder, L. J. M. (1987). The continuous flow model revisited: Perceptual and motor aspects. In R. Johnson, Jr., J. W. Rohrbaugh, & R. Parasuraman (Eds.), *Current trends in event related potential research* (EEG Suppl. 40, pp. 270-278). Amsterdam: Elsevier.

Smid, H. G., Mulder, G., & Mulder, L. J. (1990). Selective response activation can begin before stimulus recognition is complete: A psychophysiological and error analysis of continuous flow. *Acta psychologica*, *74*(2-3), 169-210.

Spapé, M. M., Band, G. P., & Hommel, B. (2011). Compatibility-sequence effects in the Simon task reflect episodic retrieval but not conflict adaptation: Evidence from LRP and N2. *Biological Psychology*, *88*(1), 116-123.

Stins, J. F., Polderman, J. C., Boomsma, D. I., & de Geus, E. J. C. (2005). Response interference and working memeory in 12-year-old children. *Child Neurospychology, 11*, 191-201.

Stoffels, E. J. (1996a). On stage robustness and response selection routes: Further evidence. *Acta Psychologica, 91*, 67-88.

Stoffels, E. J. (1996b). Uncertainty and processing routes in the selection of a response: An S-R compatibility study. *Acta Psychologica, 94*, 227-252.

Stoffels, E. J., & Van der Molen, M. W. (1988). Effects of visual and auditory noise on visual choice reaction time in a continuous-flow paradigm. *Perception & Psychophysics*, *44*(1), 7-14.

Stoffels, E. J., van der Molen, M. W., & Keuss, P. J. G. (1989). An additive factors analysis of the effects of location cues associated with auditory stimuli on stages of information processing. *Acta Psychologica, 70*, 161-197.

Stürmer, B., & Leuthold, H. (2003). Control over response priming in visuo-motor processing: a lateralized event-related potential study*. Experimental Brain Research, 153*, 35-44.

Stürmer, B., Leuthold, H., Soetens, E., Schröter, H., & Sommer, W. (2002). Control over location-based response activation in the Simon task: behavioral and electrophysiological evidence. *Journal of Experimental Psychology: Human Perception and Performance*, *28*(6), 1345.

Stürmer, B., Redlich, I., Irlbacher, K., & Brandt, S. (2007). Executive control over response priming and conflict: a transcranial magnetic stimulation study. *Experimental Brain Research, 183,* 329-339.

Treccani, B., Umiltà, C., & Tagliabue, M. (2006). Simon effect with and without awareness of the accessory stimulus. *Journal of experimental psychology: human perception and performance, 32(2)*, 268.

Turken, A. U., & Swick, D. (1999). Response selection in the human anterior cingulate cortex. *Nature Neuroscience, 2*, 920-924.

Valle- Inclán, F. (1996). The locus of interference in the Simon effect: An ERP study. *Biological Psychology, 43*, 147-162.

Valle-Inclán, F. (1996). The Simon effect and its reversal studied with event-related potentials. *International Journal of Psychophysiology*, *23*(1), 41-53.

Vallesi, A., Mapelli, D., Schiff, S., Amodio, P., & Umiltà, C. (2005). Horizontal and vertical Simon effect: different underlying mechanisms? *Cognition, 96*, B33-B43.

van 't Ent, D. (2002). Perceptual and motor contributions to performance and ERP components in a flanker reaction task. *Clinical Neurophysiology, 113*, 270-283.

van Boxtel, G. J. M., van der Molen, M. W., Jennings, J. R., & Brunia, C. H. M. (2001). A psychophysiological analysis of inhibitory motor control in the stop-signal paradigm. *Biological Psychology, 58*, 229-262.

van Boxtel, G. J. M., van der Molen, M. W., Jennings, R., & Brunia, C. H. M. (2001). A psychophysiological analysis of inhibitory control in the stop-signal paradigm. Biological Psychology, 58, 229-262.

van de Laar, M. C., van den Wildenberg, W. P., van Boxtel, G. J., Huizenga, H. M., & van der Molen, M. W. (2012). Lifespan changes in motor activation and inhibition during choice reactions: a Laplacian ERP study. *Biological psychology, 89*(2), 323-334.

van de Laar, M. C., van den Wildenberg, W. P., van Boxtel, G. J., & van der Molen, M. W. (2014). Development of response activation and inhibition in a selective stop-signal task. *Biological psychology, 102*, 54-67.

van den Wildenberg, W. P. M., & van der Molen, M. W. (2004). Additive factors analysis of inhibitory processing in the stop-signal paradigm. *Brain & Cognition, 56*, 253-266.

van der Lubbe, R. H. J., Jaśkowski, P., Wauschkuhn, B., & Verleger, R. (2001). Influence of time-pressure in a simple response task, a choice-by-location task, and the Simon task. *Journal of Psychophysiology 15*, 241-255.

van Duren, L. L., & Sanders, A. F. (1988). On the robustness of the additive factors stage structure in blocked and mixed designs. *Acta Psychologica, 69*, 83-94.

van Veen, V., & Carter, C. S. (2002). The timing of action-monitoring processes in the anterior cingulate cortex. *Journal of Cognitive Neuroscience, 14*, 593-602.

Vandierendonck, A., Liefooghe, B., & Verbruggen, F. (2010). Task switching: interplay of reconfiguration and interference control. *Psychological bulletin*, *136*(4), 601.

vel Grajewska, B. Ż., Sim, E. J., Hoenig, K., Herrnberger, B., & Kiefer, M. (2011). Mechanisms underlying flexible adaptation of cognitive control: Behavioral and neuroimaging evidence in a flanker task. *Brain research, 1421*, 52-65.

Verbruggen, F. (2016). Executive control of actions across time and space. *Current directions in psychological science, 25*(6), 399-404.

Verguts, T., & Notebaert, W. (2009). Adaptation by binding: a learning account of cognitive control. *Trends in cognitive sciences, 13*(6), 252-257.

Verleger, R. (1997). On the utility of P3 latency as an index of mental chronometry. *Psychophysiology, 34*, 131-156.

Vidal, F., Grapperon, J., Bonnet, M., & Hasbroucq, T. (2003). The nature of unilateral motor commands in between-hand choice tasks as revealed by surface Laplacian estimation. *Psychophysiology, 40*, 796-805.

Vidal, F., Hasbroucq, T., Grapperon, J., & Bonnet, M. (2000). Is the 'error negativity' specific to errors? *Biological Psychology, 51*, 109-128.

Vogel, E. K., & Luck, S. J. (2000). The visual N1 component as an index of a discrimination process. *Psychophysiology, 37(02)*, 190-203.

Von Gunten, C. D., Volpert‐Esmond, H. I., & Bartholow, B. D. (2018). Temporal dynamics of reactive cognitive control as revealed by event‐related brain potentials. *Psychophysiology, 55*(3), e13007.

Vu, K. P. L., & Proctor, R. W. (2001). Determinants of right–left and top–bottom prevalence for two-dimensional spatial compatibility. *Journal of Experimental Psychology: Human Perception and Performance*, *27*(4), 813.

Vu, K.-P. L., & Proctor, R. W. (2004). Mixing compatible and incompatible mappings: Elimination, reduction, and enhancement of spatial compatibility effects. *Quarterly Journal of Experimental Psychology, 57A* (3), 539-556.

Wascher, E. (2005). The timing of stimulus localization and the Simon effect: an ERP study. *Experimental Brain Research, 163*, 430-439.

Wascher, E. & Wauschkuhn, B. (1996). The interaction of stimulus- and response-related processes measured by event-related lateralizations of the EEG. *Electroencaphology and Clinical Neurophysiology, 99*, 149-162.

Wascher, E., Reinhard, M., Wauschkuhn, B., & Verleger, R. (1999). Spatial S-R compatibility with centrally presented stimuli. An event-related asymmetry study on dimensional overlap. *Journal of Cognitive Neuroscience, 11*, 214-229.

Wendt, M., Heldmann, M., Münte, T. F., & Kluwe, R. H. (2007). Disentangling sequential effects of stimulus-and response-related conflict and stimulus-response repetition using brain potentials. *Journal of Cognitive Neuroscience, 19*(7), 1104-1112.

Wendt, M., Kluwe, R. H., & Peters, A. (2006). Sequential modulations of interference evoked by processing task-irrelevant stimulus features. Journal of Experimental Psychology: *Human Perception and Performance, 32*, 644-667.

Wiegand, K., & Wascher, E. (2005). Dynamic aspects of stimulus-response correspondence: evidence for two mechanisms involved in the Simon effect. *Journal of Experimental Psychology: Human Perception and Performance, 31*, 453-464.

Willemssen, R., Hoormann, J., Hohnsbein, J., & Falkenstein, M. (2004). Central and parietal event-related lateralizations in a flanker task. *Psychophysiology, 41*, 762-771.

Woodworth, R. S. (1938). *Experimental psychology*. New York: Holt, Rinehart and Winston.

Wylie, G. & Allport, A. (2000). Task-switching and the measurement of "switch-costs". *Psychological Research, 63,* 212-233.

Yeung, N., & Monsell, S. (2003). Switching between tasks of unequal familiarity: The role of stimulus-attribute and response-set selection. *Journal of Experimental Psychology: Human perception and performance, 29*(2), 455.

Yeung, N., Bogacz, R., Holroyd, C. B., & Cohen, J. D. (2004). Detection of synchronized oscillations in the electroencephalogram: an evaluation of methods. *Psychophysiology, 41*(6), 822-832.

Yeung, N., Botvinick, M. M., & Cohen, J. D. (2004). The neural basis of error detection: Conflict monitoring and the error-related negativity. *Psychological Review, 111*, 931–959.

Yordanova, J., Kolev, V., Hohnsbein, J., & Falkenstein, M. (2004). Sensorimotor slowing with ageing is mediated by a functional dysregulation of motor- generation processes: Evidence from high-resolution event-related potentials. *Brain, 127*, 351–362

Zhang, J., & Kornblum, S. (1997). Distributional analysis and De Jong, Liang, and Lauber's (1994) dual-process model of the Simon effect. *Journal of Experimental Psychology: Human Perception and Performance, 23(5)*, 1543.

Zorzi, M., Mapelli, D., Rusconi, E., & Umiltà, C. (2003). Automatic spatial coding of perceived gaze direction is revealed by the Simon effect. *Psychonomic Bulletin & Review, 10(2)*, 423-429.

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Lay Summary

At work, at home, and on the road, people are more and more often required to make fast decisions and give immediate responses, making it essential to understand the factors that influence human performance. This work investigated how human beings use cognitive control in increasingly complex tasks, aiming to understand how the brain responds in predictable and unpredictable situations. Scientists have developed several techniques for investigating such cognitive functions, and this work combined behavioural and electrophysiological methods: stimulus-response compatibility and EEG. Stimulus-response compatibility (SRC) concerns the relationship between stimuli (such as a red traffic light) and responses (such as pressing the brake), and especially the ease with which a particular stimulus lends to a particular response. We can manipulate SRC, and thus task complexity, by designing tasks with different combinations of stimuli and responses. The effects of task complexity on the brain can then be detected in EEG (electrophysiological measures recorded from the scalp) during experimental tasks. Event Related Potentials (ERPs) offer a means to investigate the precise timing of interference (with more difficult tasks/SRC) and potential measures of cognitive control.

The results of four experiments revealed how cognitive control can reduce or increase interference associated with SRC and task difficulty, depending on response strategies and on how predictable a specific type of interference is. Chapters 2 and 3 compared interference induced by the location of the stimulus or by additional 'distractor' stimuli, and performance and ERP results suggested that resolving each type of interference relies on different strategies. Chapters 4 and 5 demonstrate how people can reduce interference using proactive (preparatory) control, but how preparing for the most likely or most difficult task can lead to performance detriments and late correction (reactive control) on the unprepared task. In other words, control strategies seem to play an essential role in determining how quickly and accurately we can respond to changing task demands. However, the final discussion relates the experiments to more recent studies, theories and computational modeling, concluding that multiple strategies could still be accountable to a general control mechanism that is most effective with constant updating.